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TRANSACTIONS
OF
THE MEDICAL SOCIETY
OF
LONDON.

VOLUME THE FIFTEENTH.



EDITED BY

A. MARMADUKE SHEILD, M.B., F.R.C.S.,

AND

WILLIAM PASTEUR, M.D.

LONDON:


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THE present volume comprises the Transactions of the Society during its one hundred and nineteenth Session, from October 19th, 1891, to May 2nd, 1892.



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| 1780. JOHN KOOYSTRA, M.D. | 1823. EDWARD GRAINGER. |
| 1781. SAMUEL FOART SIMMONS,
M.D., F.R.S. | 1824. GORDON SMITH, M.D. |
| 1782. LOFTUS WOOD, M.D. | 1825. EUSEBIUS ARTHUR LLOYD. |
| 1783. JOHN SIMS, M.D. | 1826. JOHN HASLAM, M.D. |
| 1784. JOHN WHITEHEAD, M.D. | 1827. WILLIAM KINGDOM. |
| 1785. JOHN RELPH, M.D. | 1828. JOHN BURNE, M.D. |
| 1787. JOSEPH HOOPER. | 1829. WILLIAM GREVILLE JONES. |
| 1788. JOHN MEYER, M.D. | 1830. LEONARD STEWART, M.D. |
| 1789. RICHARD DENNISON, M.D. | 1831. MONTAGUE GOSSETT. |
| 1790. GEORGE WALLIS, M.D. | 1832. JOHN WHITING, M.D. |
| 1791. SAMUEL SUTTON, M.D. | 1833. FREDERICK SALMON. |
| 1792. EDWARD FRYER, M.D. | 1834. WILLIAM SHEARMAN, M.D. |
| 1793. JAMES JAMESON, M.D. | 1835. WALTER COOPER DENDY. |
| 1794. GILBERT THOMPSON, M.D. | 1836. WILLIAM F. BLICKE, M.D. |
| 1795. JOHN ABERNETHY. | 1837. EDWARD HEADLAND. |
| 1796. JOHN COAKLEY LETTSOM,
M.D., F.R.S. | 1838. THEOPHILUS THOMPSON, M.D.,
F.R.S. |
| 1797. JAMES WARE. | 1839. GEORGE PILCHER. |
| 1798. SAMUEL FERRIS, M.D., F.R.S. | 1840. JAMES RISDON BENNETT, M.D. |
| 1799. EDWARD FORD. | 1841. WM. DINGLE CHOWNE, M.D. |
| 1800. THOMAS BRADLEY, M.D. | 1842. HENRY HANCOCK. |
| 1801. WILLIAM CHAMBERLAINE. | 1843. LEONARD STEWART, M.D. |
| 1802. JOHN SIMS, M.D. | 1844. THOMAS BELL, F.R.S. |
| 1803. JOHN ANDRÉE. | 1845. MARSHALL HALL, M.D. |
| 1804. JOHN COAKLEY LETTSOM,
M.D., F.R.S. | 1846. JOHN BISHOP, F.R.S. |
| 1805. GEORGE PINCKHARD, M.D. | 1847. GOLDING BIRD, M.D., F.R.S. |
| 1806. HENRY FIELD. | 1848. FRANCIS HIRD. |
| 1807. JOSEPH ADAMS, M.D. | 1849. WILLIAM HUGHES WILL-
SHIRE, M.D. |
| 1808. JOHN MASON GOOD, F.R.S. | 1850. FRANCIS HIRD. |
| 1809. SAYER WALKER, M.D. | 1851. RICHARD ROWLAND. |
| 1810. GEORGE BIRKBECK, M.D. | 1852. EDWIN CANTON. |
| 1811. WILLIAM BLAIR. | 1853. JOHN SNOW, M.D. |
| 1812. RICHARD TEMPLE, M.D. | 1854. HENRY SMITH. |
| 1813. RICHARD SAUMAREZ, F.R.S. | 1855. JAMES FERNANDEZ CLARKE. |
| 1814. GEORGE REES, M.D. | 1856. BENJ. WARD RICHARDSON,
M.D., F.R.S. |
| 1815. JOHN TAUNTON. | 1857. WILLIAM ADAMS. |
| 1816. HENRY CLUTTERBUCK, M.D. | 1858. ALFRED BARING GARROD, M.D. |
| 1817. JAMES STEVENSON. | 1859. CHARLES HENRY FELIX ROUTH,
M.D. |

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| 1860. JOHN GAY. | 1878. ALFRED CARPENTER, M.D. |
| 1861. ARTHUR LEARED, M.D. | 1879. WALTER JOHN COULSON. |
| 1862. VICTOR DE MÉRIC. | 1880. WILLIAM HENRY BROADBENT,
M.D. |
| 1863. SAMUEL OSBORNE HABERSHON,
M.D. | 1881. ARTHUR EDWARD DURHAM. |
| 1864. JOHN LOUIS WILLIAM THUDICHUM, M.D. | 1882. EDMUND SYMES THOMPSON,
M.D. |
| 1865. ROBERT GREENHALGH, M.D. | 1883. EDWARD LUND. |
| 1866. THOMAS CHRISTOPHER WEEDEN COOKE. | 1884. CHARLES THEODORE WILLIAMS,
M.D. |
| 1867. FREDERICK WILLIAM HEADLAND, M.D. | 1885. GEORGE MURRAY HUMPHRY,
M.D., F.R.S. |
| 1868. WILLIAM FREDERICK TEEVAN. | 1886. RICHARD DOUGLAS POWELL,
M.D. |
| 1869. GEORGE DUNCAN GIBB, M.D. | 1887. SIR WILLIAM MACCORMAC,
F.R.C.S. |
| 1870. FRANCIS MASON. | 1888. SIR JOSEPH FAYRER, K.C.S.I.,
M.D., F.R.S. |
| 1871. WILLIAM CHOLMELEY, M.D. | 1889. JONATHAN HUTCHINSON, F.R.S. |
| 1872. FREDERICK JAMES GANT. | 1890. ARTHUR ERNEST SANSOM, M.D. |
| 1873. JOHN COCKLE, M.D. | 1891. SIR JOSEPH LISTER, Bart.,
F.R.S. |
| 1874. ROBERT BRUDENELL CARTER. | 1892. SIR JAMES CRICHTON BROWNE,
M.D., F.R.S. |
| 1875. GEORGE BUCHANAN, M.D. | |
| 1876. ERASMUS WILSON, F.R.S. | |
| 1877. JOHN HUGHLINGS JACKSON,
M.D., F.R.S. | |

THE FOTHERGILLIAN GOLD MEDALLISTS.

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|---------------------------------------|---|
| 1787. WILLIAM FALCONER, M.D. | 1851. RICHARD HODGES. |
| 1790. ROBERT WILLAN, M.D. | 1852. FREDERICK WILLIAM HEADLAND. |
| 1791. JOHN COAKLEY LETTSOM, M.D. | 1853. ALFRED WILLIAM POLAND. |
| 1795. JOHN MASON GOOD. | 1854. BENJAMIN WARD RICHARDSON,
M.D. |
| 1801. FRANCIS BOUTTATZ, M.D. | 1856. WILLIAM BURKE RYAN. |
| 1803. EDWARD JENNER, M.D. | 1857. EDWIN CANTON. |
| 1824. ROBERT W. BAMPFIELD. | 1858. THOMAS HERBERT BARKER,
M.D. |
| 1828. JOHN GEORGE PARRY. | 1859. ALDERMAN THOMAS HOUGHTON
WATERS. |
| 1831. WILLIAM AUGUSTUS GUY. | 1868. JOHN CLAY. |
| 1834. WILLIAM JAMES CLEMENT. | 1870. THOS. SMITH CLOUSTON, M.D. |
| 1835. GEORGE MOORE. | 1872. EDWARDS CRISP, M.D. |
| 1836. THOMAS EGERTON BRYANT. | 1873. JOHN KENT SPENDER, M.D. |
| 1838. GEORGE PILCHER. | 1877. PETER MURRAY BRAIDWOOD,
M.D. |
| 1840. SAMUEL OSBORN. | 1878. JOHN MILNER FOTHERGILL,
M.D. |
| 1842. JAMES RISDON BENNETT, M.D. | 1882. THOMAS MICHAEL DOLAN, M.D. |
| 1843. JOHN WEAVER LEVER, M.D. | 1883. NORMAN PORRITT. |
| 1844. HENRY PRATT ROBARTS. | 1886. JOHN STRAHAN. |
| 1845. WALTER COOPER DENDY. | 1888. HOBART AMORY HARE, M.D.,
U.S.A. |
| 1846. ROBERT MORTIMER GLOVER,
M.D. | |
| 1847. SILAS STEDMAN. | |
| 1849. JOHN MILLIGAN. | |
| 1850. RICHARD PAYNE COTTON, M.D. | |

THE HONORARY FELLOWS.

-
1876. BARNES, J. K., M.D., Surgeon-General U.S. Army, Washington.
1881. BILLINGS, JOHN S., M.D., Washington, Surgeon to the United States Army; Librarian to the Surgeon-General's Library, Washington.
1881. BILLROTH, THEODORE, M.D., Professor of Surgery in the University of Vienna.
1881. CHARCOT, Professor J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor of the Faculty of Medicine, Paris.
1873. CHAUVEAU, A., Professor of Physiology at the Medical School of Lyons.
1890. CRUDELI, TOMMASI, M.D., Rome.
1881. DA COSTA, J. M., M.D., Professor of Medicine in the Jefferson Medical College, 1700, Walnut-street, Philadelphia.
1881. EMMET, THOMAS ADDIS, M.D., 89, Madison-avenue, Surgeon to the Woman's Hospital of the State of New York.
1886. GAIRDNER, WILLIAM TENNANT, M.D., LL.D. Edin., F.R.C.P. Edin.
1881. HALLA, JOSEPH, Professor of Medicine in the University of Prague.
1869. HARE, CHARLES JOHN, M.D., Berkeley House, Manchester-square, W., Consulting Physician to University College Hospital, and late Professor of Clinical Medicine in University College. P, VP 2. C 8, LL. *Trustee.*
1873. HELMHOLTZ, HERMANN LUDWIG FERDINAND, M.D., Professor of Physics and Physiological Optics in the University of Berlin.
1890. HOLMGREN, FRITHIOF, Professor, Upsala.
1883. HUMPHRY, Sir GEORGE MURRAY, M.D., F.R.S., Professor of Surgery in the University of Cambridge. O, C 2.
1873. HUXLEY, The Right Honourable THOMAS HENRY, LL.D., F.R.S., Hodeslea, Eastbourne, Professor of Biology in the Normal School of Science and in the Royal School of Mines.
1875. JENNER, Sir WILLIAM, Bart., K.C.B., D.C.L., LL.D., M.D., F.R.S., Greenwood, Durley, Hants, Physician-in-Ordinary to H.M. the Queen and to H.R.H. the Prince of Wales; late President of the Royal College of Physicians; Emeritus Professor of Clinical Medicine in University College, London; Consulting Physician to University College Hospital.
1890. KOCHER, THEODOR, Professor, Berne.
1884. LARREY, Baron, M.D., Paris, Rue de Lille, 91.
1883. LE ROY DE MERICOURT, A., M.D., Paris.

- 1890. LOMBARD, HENRI-CLERMOND, M.D., Geneva.
- 1878. MITCHELL, S. WEIR, M.D., Walnut-street, Philadelphia.
- 1881. NUSSBAUM, JOHN NEPOMUK RITTER VON, M.D., Professor of Surgery in the University of Munich.
- 1875. OLLIER, Professor, Lyons.
- 1873. OWEN, Sir RICHARD, K.C.B., F.R.S., Sheen Lodge, Richmond Park, Superintendent of the Natural History Department of the British Museum.
- 1873. PAGET, Sir JAMES, Bart., D.C.L., LL.D., F.R.S., 1, Harewood-place, Hanover-square, W., Serjeant-Surgeon to H.M. the Queen; Surgeon to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital.
- 1876. PANCOAST, JOSEPH, M.D., 1030, Chesnut-street, Philadelphia, Professor of Anatomy in the Jefferson Medical College.
- 1890. PASTEUR, LOUIS, Member of the Institute of France, Paris.
- 1877. SANNÉ, A., 12, Place de Laborde, Paris.
- 1835. SEAONE, M., M.D., Salamanca.
- 1881. TARNIER, STEPHANIE, M.D., Professor of Obstetric Medicine in the School of Medicine, Paris.
- 1873. TYNDALL, JOHN, F.R.S.
- 1881. VERNEUIL, AUGUSTE ARISTIDE, M.D., Professor of Medicine in the School of Medicine, Paris.
- 1873. VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin.

CORRESPONDING FELLOWS.

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1851. ALBARO, J. MENDEZ, Madrid.
 1882. BADALONI, GIUSEPPE, M.D., Fano, Prov. di Pesaro, Italy.
 1856. BAKER, ALBERT, M.D., The Laurels, Pinhoe, Exeter.
 1855. BEARDSLEY, AMOS, Bay Villa, Grange, Lancashire.
 1850. BENAVENTE, MARIANO, Madrid.
 BENEKE, F. W., M.D., New York.
 1850. BÖHM, PROFESSOR, M.D., Vienna.
 BOTTINI, GIUSEPPE, M.D., Milan.
 1837. BUHRING, J. J., M.D., Berlin.
 1874. BURNES, ALEXANDER GEORGE, M.D., Port Elizabeth, Cape of Good
 Hope.
 CADE, THOMAS CHARLES, Spondon, Derby.
 1855. COATES, CHARLES, M.D., F.R.C.P., 10, Circus, Bath, Consulting Physician
 to the Bath Royal United Hospital. c 3.
 1850. COX, WILLIAM ISIDORE, Hawkesbury, Upton, Gloucestershire. c.
 1876. DAWES, RICHARD, St. Mark, Gawler, South Australia.
 DE MUYNCK, J., M.D., Ghent.
 1836. ECSTEIN, SIGISMUND, M.D., Vienna.
 EYLANDT, JOHANN EMIL, M.D., Curland, Russia.
 1853. FALLOT, R., M.D., St. Laurent d'Aigouze, Montpellier, France.
 1889. FRANK, PHILIP, M.D., F.R.C.P., Cannes, France.
 1876. GRIFFITH, RICHARD GLYN, Allahabad, India.
 1864. HASENFELD, EMMANUEL, M.D., Pesth.
 HYMAN, —, M.D., Antwerp.
 1851. IZGUIERDO, SEBASTIAN OBTEGA, Madrid.
 1875. JONES, PHILIP SYDNEY, M.D., F.R.C.S., Examiner in Medicine in the
 University of Sydney, Australia, Hon. Consulting Surgeon to the
 Sydney Infirmary.
 1861. JOURNEZ, HENRI, M.D., 43, Rue de la Charité, Bruxelles, Belgique.
 1851. KÖLLIKER, ALBERT, M.D., Professor of Anatomy and Physiology at the
 University of Wurzburg.
 1876. LEIGHTON, WALTER H., M.D., Lowell, Massachusetts, U.S.
 LEON, JOSE, Madrid.
 1851. LLANOS, ANTONIO CAMPO, Madrid.
 1851. LOVERA, JOSE, Madrid.

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1851. MARINO, BONIFACIO MATREOS, Madrid.
MENDEZ, BARTHOLOME, Madrid.
MOLINA, M. M., Madrid.
NEGRI, GAETANO, M.D., Pisa.
ORTEGA, J. R., Madrid.
1865. PERUZZI, DOMENICO, M.D., 22, Via Mazzini, Bologna.
1882. RESTREPO, ALESSANDO EDUARDO, M.D., Medellin, Columbia, U.S.A.
1886. ROCHA, A., M.D., Coimbra, Beira, Portugal.
1860. ROUSSEL, M.D., Dean of the Faculty of Medicine, Montpellier.
SCHARLAN, GUS. W., M.D., Stettin, Prussia.
1876. SCHMITZ, RICHARD, M.D., Neuenahr.
1874. SCHUTGOWSKY, J., St. Petersburg.
1851. SESSE, M., Mesqui, Madrid.
STOCKWELL, THOMAS GOLDESBOUGH, F.R.C.S., 6, Circus, Bath,
Surgeon to the Bath Royal United Hospital.
TEREZA, FELIX GARCIA, Madrid.
VALDEZ, FRANCO CORTIGO, Madrid.
WILLIAMS, CHARLES, F.R.C.S. Edin., 48, Prince of Wales-road,
Norwich ; Surgeon to the Norfolk and Norwich Hospital.

THE FELLOWS

OF

THE MEDICAL SOCIETY OF LONDON.

EXPLANATION OF ABBREVIATIONS.

P.—PRESIDENT.	FM.—FOTHERGILLIAN GOLD MEDALLIST.
VP.—VICE-PRESIDENT.	SM.—SILVER MEDALLIST.
T.—TREASURER.	O.—ORATOR.
L.—LIBRARIAN.	CFC.—CHAIRMAN, HOUSE AND FINANCE COMMITTEE.
S.—SECRETARY.	§—SEC. FOR FOREIGN CORRESPONDENCE.
C.—COUNCILLOR.	LL.—LETT SOMIAN LECTURER. *—LIFE MEMBERS.
TR.—TRUSTEE.	

The number prefixed signifies the date of election. The figures appended indicate the number of Sessions served, and refer to past appointments ONLY.

1890. ABBOT-ANDERSON, WILLIAM MAURICE, M.B., 10, Old Cavendish-street, Cavendish-square, W.
1888. ABBOTT, CHARLES EDWARD, M.R.C.S., Noel House, Braintree, Essex.
1891. ABRAHAM, PHINEAS S., M.D., 2, Henrietta-street, Cavendish-square, W.
1890. ACKLAND, ROBERT CRAIG, M.R.C.S., 13, Savile-row, W.
1883. ACLAND, THEODORE DYKE, M.D., 74, Brook-street, Grosvenor-square, W.
C.
1884. ADAM, JAMES, M.D., Malling-place, West Malling, Kent.
1889. ADAMS, JAMES, M.D., 4, Chiswick-place, Eastbourne.
1878. ADAMS, JOSIAH OAKE, M.D., Brook House, Upper Clapton, E.
1852. *ADAMS, WILLIAM, F.R.C.S., 5, Henrietta-street, Cavendish-square, W.
P, C 8, O, VP 3, LL.
1878. *ALLCHIN, WILLIAM HENRY, M.B., F.R.S.E., 5, Chandos-street, Cavendish-square, W. *Vice-President, Hon. Librarian.*
1873. ALLEN, HENRY MARCUS, F.R.C.P. Edin., 20, Regency-square, Brighton.
1873. ALLFREY, CHARLES HENRY, M.D., Plas Newydd, St. Leonards-on-Sea.
1883. ALLINGHAM, HERBERT W., F.R.C.S., 25, Grosvenor-street, W. c 3.
1872. *ALLINGHAM, WILLIAM, F.R.C.S., 25, Grosvenor-street, W. c.
1860. ALTHAUS, JULIUS, M.D., 48, Harley-street, W. c 5, § 3.

1887. ANDERSON, JAMES, M.D., 41, Wimpole-street, W.
 1885. ANDERSON, JOHN, M.D., C.I.E., 9, Harley-street, W.
 1889. ANDERSON, WILLIAM, F.R.C.S., 2, Harley-street, W.
 1888. ANDREWES, FREDERICK WILLIAM, M.B., 15, Upper Brook-street, W.
 1869. ARMITAGE, SAMUEL HARRIS TATHAM, M.D., 39, Grosvenor-street, W.
 1873. ATKINSON, EDWARD, M.R.C.S., 93, Albion-street, Leeds.
 1872. AVELING, JAMES HOBSON, M.D., 1, Upper Wimpole-street, W.
 1892. AYRES, CHARLES JAMES, M.D., 15, Grosvenor-road, Westminster, S.W.
1873. BAGSHAW, FREDERIC, M.D., 5, Warrior-square, St. Leonards-on-Sea. c.
 1871. BAILEY, GEORGE HEWLETT, M.R.C.S., 9, Cavendish-place, W.
 1892. BAILEY, HENRY FREDERICK, M.R.C.S., The Hollies, Lee-terrace, Lee, S.E.
 1891. BAILY, PERCY J., M.B., County Asylum, Hanwell, W.
 1876. *BAKER, HENRY FRANCIS, F.R.C.S. Edin., 2, Mandeville-place, Manchester-square, W. c.
 1890. BAKER, WILLIAM HENRY, M.R.C.S., 40, Norfolk-terrace, Bayswater, W.
 1891. BALL, JAMES BARRY, M.D., 54, Wimpole-street, W.
 1881. BALLANCE, CHARLES ALFRED, M.S., 56, Harley-street, W. s 2, c.
Councillor.
 1884. BANKS, W. MITCHELL, F.R.C.S., 38, Rodney-street, Liverpool. *Orator.*
 1859. BARNES, JOHN WICKHAM, F.R.C.S., 3, Bolt-court, E.C. s 2, VP, c 3.
 1883. BARNES, ROBERT, M.D., 7, Queen Anne-street, W. LL, c.
 1874. BARRETT, HOWARD, M.R.C.S., 3, Tavistock-square, W.C.
 1884. BARROW, ALBERT BOYCE, F.R.C.S., 37, Wimpole-street, W. *Councillor.*
 1886. BARWELL, RICHARD, F.R.C.S., 55, Wimpole-street, W.
 1884. BATEMAN, FREDERICK AUGUSTUS NEWTON, M.R.C.S., 4, Charles-street, St. James's-street, S.W.
 1886. BATTERHAM, JOHN WILLIAMS, M.B., Bank House, Grand-parade, St. Leonards-on-Sea.
 1888. BATTLE, WILLIAM HENRY, F.R.C.S., 6, Harley-street, W.
 1882. BEACH, FLETCHER, M.B., Darenth Asylum, Dartford, Kent. c.
 1887. BEALE, EDWIN CLIFFORD, M.B., 23, Upper Berkeley-street, W.
 1891. BEALE, PEYTON T. B., F.R.C.S., 61, Grosvenor-street, W.
 1880. BEEVOR, CHARLES EDWARD, M.D., 33, Harley-street, W. s 2, c.
Trustee.
 1889. BEEVOR, Sir HUGH REEVE, Bart., M.B., King's College-chambers, Strand, W.C.
 1887. BENHAM, FREDERICK LUCAS, M.D., 93, Elizabeth-street, Eaton-square, S.W.
 1881. BENNET, ROBERT OTTIWELL-GIFFORD, M.D., Tankerville House, Park-place, Buxton.
 1883. BENNETT, WILLIAM HENRY, F.R.C.S., 1, Chesterfield-street, Mayfair, W.
 1887. BERRY, JAMES, F.R.C.S., 60, Welbeck-street, W.
 1873. BEVERIDGE, JAMES SPOWART, M.R.C.P. Edin. (address uncommunicated).

1890. BIDWELL, LEONARD ARTHUR, F.R.C.S., 21, Bentinck-street, Manchester-square, W.
1868. BIRD, GEORGE, M.D., 49, Welbeck-street.
1888. BIRD, MATTHEW MITCHELL, M.D., St. Mary's Hospital, W.
1850. *BIRKETT, JOHN, F.R.C.S., 62, Green-street, Grosvenor-square, W. VP, C 6.
1883. BISS, CECIL YATES, M.D., 135, Harley-street, W.
1889. BISSHOPP, FRANCIS ROBERT BRYANT, M.B., Belvedere, Lonsdale-gardens, Tunbridge Wells.
1886. *BLACK, WILLIAM GALL, F.R.C.S., 2, George-square, Edinburgh.
1885. BLAKE, JOHN FRENCH, Terrace House, Camberwell-green, S.E.
1881. BLAKER, WALTER CAMPBELL, Bognor, Sussex.
1888. BLANC, LEON, M.D., Aix les Bains, France.
1871. *BLOXAM, JOHN ASTLEY, F.R.C.S., 8, George-street, Hanover-square, W. V.P 2, s 2, c 3.
1867. BOND, THOMAS, F.R.C.S., 7, The Sanctuary, Westminster, S.W. c.
1879. BOTT, HENRY, M.R.C.S., Brentford, Middlesex.
1872. BOULTON, PERCY, M.D., 6, Seymour-street, Portman-square, W. c.
1886. BOURNS, NEWCOME WHITELAW, M.D., 449, Fulham-road, S.W.
1886. BOUSTEAD, ROBINSON, M.D. (Brigade Surgeon), 10, Palmeira-avenue, West Brighton.
1889. BOWLES, ROBERT LEAMON, M.D., 8, West-terrace, Folkestone.
1883. BRADSHAW, JAMES DIXON, M.B., 30, George-street, Hanover-square, W.
1868. BRAIDWOOD, PETER MURRAY, M.D., 11, Grosvenor-gardens, Willesden-green, N.W. F.M., 1877.
1869. BRAINE, FRANCIS WOODHOUSE, F.R.C.S., 56, Maddox-street, W. VP 2, s 2, c 3, SM.
1889. BRAINE, C. CARTER, F.R.C.S., 56, Maddox-street, W.
1876. BREWER, ALEXANDER HAMPTON, 136, Richmond-road, Dalston, E.
1873. BRIDGWATER, THOMAS, M.B., Harrow, Middlesex. *Councillor*.
1887. BRISTOWE, JOHN SYER, M.D., F.R.S., 13, Old Burlington-street, W. *Lettsomian Lecturer*.
1862. BROADBENT, WILLIAM HENRY, M.D., 34, Seymour-street, Portman-square, W. P, VP, O, LL, C 4.
1890. BROOK, WILLIAM FREDERICK, F.R.C.S., Pennard House, Walters-road, Swansea.
1879. BROOKFIELD, JOHN STORRS, M.D., 2, Devonshire-villas, Brondesbury, N.W.
1878. BROOKS, JOB EDWIN, 54, Mill-street, Ludlow, Salop.
1878. BROWN, ANDREW, M.D., Elton Villa, 1, Bartholomew-road, Kentish Town, N.W.
1871. BROWN, JOHN, Belmont Lodge, St. John's-hill, New Wandsworth, S.W.
1889. BROWNE, GEORGE BUCKSTON, M.R.C.S., 80, Wimpole-street, W.
1871. BROWNE, Sir JAMES CRICHTON, M.D., F.R.S., Queen Anne-mansions, Westminster, S.W. O, C.

1873. BROWNE, LENNOX, F.R.C.S. Edin., 15, Mansfield-street, Portland Place, W.
1887. BRUCE, JOHN MITCHELL, M.D., 70, Harley-street, W.
1873. BRUNJES, MARTIN, M.R.C.S., 33A, Gloucester-place, Bryanston-square, W.
1862. BRUNTON, JOHN, M.D., 21, Euston-road, N.W. VP, c 2.
1874. *BRUNTON, THOMAS LAUDER, M.D., F.R.S., 10, Stratford-place, W. LL, VP, c 4, SM.
1850. *BRYANT, THOMAS, F.R.C.S., 65, Grosvenor-street, W., President of the Royal College of Surgeons. P, VP, LL, s 2, c 4. *Trustee*.
1858. BUCHANAN, Sir GEORGE, M.D., F.R.S., 27, Woburn-square, W.C. P, LL, VP, O, c 3.
1883. BULL, WILLIAM HENRY, St. Oswald's House, Stony Stratford, Bucks.
1885. *BUNNY, J. BRICE, M.R.C.S., Newbury, Berks.
1872. BURGER, ALEXANDER, M.D., 49, Finsbury-square, E.C.
1890. BUTLER, PATRICK, L.K.Q.C.P., 22, Duke-street, Portland-place, W.
1886. BUTLER-SMYTHE, ALBERT CHARLES, F.R.C.S., 76, Brook-street, W.
1872. BYAS, EDWARD HEGLEY, M.R.C.S., 10, Cambridge-gate, Regent's Park, N.W.
1886. CAHILL, JOHN, F.R.C.S., 12, Seville-street, Lowndes-square, Hyde Park, S.W.
1891. CALVERT, JAMES, M.D., 36, Queen Anne-street. W.
1888. CAMPBELL, CHARLES M., M.D., 37, Queen Anne-street, W.
1890. CARDEW, HARRY WARNELL DENTON, M.R.C.S., 53, Harley-street, W.
1889. CARNALL, EDWARD, M.R.C.S., 9, Gerrard-street, W.
1882. CARPENTER, ARTHUR BRISTOWE, M.B., 34, Dingwall-road, Croydon.
1889. CARR, JOHN WALTER, M.D., 40, Bloomsbury-square, W.C.
1871. CARTER, ROBERT BRUDENELL, F.R.C.S., 27, Queen Anne-street, W., P, VP, O, LL, c 4.
1889. CARTWRIGHT, ALEXANDER, M.R.C.S., 32, Old Burlington-street, W.
1876. CARTWRIGHT, S. HAMILTON, 45, Albert-gate, Hyde-park, W.
1878. CASSIDY, JOSEPH LAMONT, M.D., 44, Harley-street, W.
1876. *CATHCART, SAMUEL, M.R.C.P. Edin., Prudhoe House, High-road, Tottenham, E.
1889. CAUTLEY, EDMUND, M.B., 15, Upper Brook-street, W.
1882. CAVAFY, JOHN, M.D., 2, Upper Berkeley-street, W. c 3.
1891. CHAPLIN, T. H. ARNOLD, M.B., 24, Finsbury-circus, E.C.
1867. CHAPMAN, JOHN, M.D., Avenue Kleber 46, Paris.
1885. CHASSEAUD, WILLIAM, M.D., Smyrna, Asia Minor.
1889. CHEYNE, WATSON, F.R.C.S., 59, Welbeck-street, W. c.
1877. *CHISHOLM, EDWIN, M.D., Sydney, New South Wales.
1871. CHURTON, THOMAS, M.D., 35, Park-square, Leeds. c.
1854. CLARK, Sir ANDREW, Bart., M.D., F.R.S., 16, Cavendish-square, W. President of the Royal College of Physicians. P, VP, LL, c 5, § 6.

1875. CLARK, ANDREW, F.R.C.S., 71, Harley-street, W.
 1873. CLARKE, THOMAS KILNER, F.R.C.S., 66, John William-street, Huddersfield.
 1883. CLARKE, WILLIAM BRUCE, F.R.C.S., 46, Harley-street, W. c.
 1879. *CLUTTON, HENRY HUGH, F.R.C.S., 2, Portland-place, W. c 2.
 1849. *COCKLE, JOHN, M.D., 5, Suffolk-place, Pall Mall, S.W. p, vp, o, l 3, c 3, sm.
 1887. COLLIER, WILLIAM, M.D., 62, High-street, Oxford.
 1883. COMPTON, FRANCIS CHARLES, 72, High-street, Poole, Dorset.
 1871. COOK, JOHN, M.D., 1, Nottingham-terrace, Regent's Park, N.W.
 1862. COOPER, ALFRED, F.R.C.S., 9, Henrietta-street, Cavendish-square, W. c 3, vp.
 1888. COOPER, ARTHUR, 20, Old Burlington-street, W.
 1872. CORFIELD, WILLIAM HENRY, M.D., 19, Savile-row. c.
 1888. COTES, CHARLES EDWARD HENRY, F.R.C.S., 20, Wilton-street, Grosvenor-place, S.W.
 1891. COUMBE, JOHN BATTEN, M.D., Wargrave, Henley-on-Thames.
 1879. COUPLAND, SIDNEY, M.D., 16, Queen Anne-street, W. c 2.
 1889. COURTNEY, GUY BUDD, M.B., 47, Seymour-street, W.
 1874. CRAIGIE, JOHN HAMILTON, 13, Savile-row, W. c.
 1873. CRAVEN, ROBERT MARTIN, F.R.C.S., J.P., 14, Albion-street, Hull.
 1889. CRAWFORD, JAMES, M.D., 4, Iddesleigh-mansions, Victoria-street, S.W.
 1881. CRIPPS, WILLIAM HARRISON, F.R.C.S., 2, Stratford-place, W. c 2.
 1880. CRITCHETT, GEORGE ANDERSON, F.R.C.S. Edin., 21, Harley-street, W.
 1880. CROCKER, HENRY RADCLIFFE, M.D., 121, Harley-street, c 3.
 1881. CROSS, FRANCIS RICHARDSON, F.R.C.S., Worcester House, Clifton, Bristol.
 1890. CULLINGWORTH, CHARLES JAMES, M.D., 46, Brook-street, W. c. *Councillor.*
 1874. CUMBERBATCH, ALPHONSO ELKIN, F.R.C.S., 17, Queen Anne-street, W. c 2.

 1892. DA COSTA, FRANCIS XAVIER, F.R.C.S., Charing Cross Hospital, W.C.
 1871. DALBY, Sir WILLIAM BARTLETT, F.R.C.S., 18, Savile-row, W. c.
 1864. DALE, GEORGE CORNELIUS, M.D., Ivy Lodge, Upper Tooting, S.W.
 1881. DALLAWAY, DENNIS JOSEPH WILLIAM, L.R.C.P. Edin., 5, Duchess-street, Portland-place, W.
 1873. DALY, OWEN, M.D., J.P., 23, Albion-street, Hull.
 1885. DAVIES-COLLEY, JOHN NEVILLE COLLEY, F.R.C.S., 36, Harley-street, W. c 3.
 1890. DAVIS, HENRY, M.R.C.S., 60, Queen Anne-street, W.
 1889. *DAVISON, JAMES, M.D., Walderslow, Bournemouth.
 1880. DAVSON, SMITH HOUSTON, M.D., Campden Villa, 203, Maida-vale, W. c 3.

1868. *DAVY, RICHARD, F.R.C.S., F.R.S.E., 33, Welbeck-street, W. VP, s 2;
SM, § 2.
1876. DAWES, H. ST. M., M.R.C.S., Gawler, South Australia.
1880. DAWSON, YELVERTON, M.D., Heathland, Southborne-on-Sea, Christ-
church, Hants.
1867. DAY, WILLIAM HENRY, M.D., 10, Manchester-square, W. c 3.
1883. DENT, CLINTON THOMAS, F.R.C.S., 61, Brook-street, Grosvenor-square,
W. c 3.
1881. DICKSON, FRANCIS KENNEDY, F.R.C.P. Edin., Wye House Lunatic
Asylum, Buxton, Derbyshire.
1891. DIVER, EBENEZER, M.D., Yately House, Kenley, Surrey.
1885. DODD, HENRY WORK, F.R.C.S., 136, Harley-street, W.
1882. DOLAN, THOMAS MICHAEL, M.D., Horton House, Halifax. FM 1882.
1881. DORAN, ALBAN HENRY GRIFFITHS, F.R.C.S., 9, Granville-place, W. c 3.
1890. DOUGLAS, WILLIAM, M.D., Dalkeith House, 7, Clarendon-place,
Leamington Spa.
1872. DOWN, JOHN LANGDON HAYDON, M.D., 81, Harley-street, W. VP, LL.
1871. DOWSE, THOMAS STRETCH, M.D., 14, Welbeck-street, W. § 3, c 3.
1877. DREW, JOHN HENRY, M.R.C.S., 48, Upper Berkeley-street, W.
c 6.
1881. DREWITT, FREDERIC GEORGE DAWTREY, M.D., 2, Manchester-square,
W.
1874. DRYSDALE, CHARLES ROBERT, M.D., 23, Sackville-street, W.
1886. DUCKWORTH, Sir DYCE, M.D., 11, Grafton-street, Piccadilly, W.
1848. *DUNCAN, JAMES, M.B., 8, Henrietta-street, Covent Garden, W.C.
1884. DUNCAN, WILLIAM, M.D., 6, Harley-street, W.
1873. *DURHAM, ARTHUR EDWARD, F.R.C.S., 82, Brook-street, W. P, o, c.
Treasurer.
1884. DURHAM, FREDERICK, F.R.C.S., 82, Brook-street, W.
1891. EASTES, THOMAS, M.D., 3, Shakespeare-terrace, Folkestone.
1860. EDMUNDS, JAMES, M.D., 29, Dover-street, W.
1880. EDWARDS, FREDERICK SWINFORD, F.R.C.S., 55, Harley-street, W.
1868. ELLIOTT, GEORGE FREDERICK, M.D., 1, Albion-street, Hull.
1882. ELLIOTT, THOMAS, M.D., Monson-place, Tunbridge Wells.
1889. EMBLETON, DENNIS CAWOOD, M.R.C.S., St. Wilfrid's, Bournemouth.
1883. EMOND, E., M.D., 113, Boulevard Beaumarchais, Paris.
1883. ENGLISH, EDGAR, M.R.C.S., High-street, Mexborough, near Rotherham.
1880. ENGLISH, THOMAS JOHNSTON, M.D., 128, Fulham-road, S.W.
1889. ESLER, ROBERT, M.D., 4, Queen's-road, Peckham, S.E.
1891. EUAN-SMITH, EUAN McLAURIN, M.R.C.S., 253, Cromwell-road, S.W.
1883. EWART, JOSEPH, M.D., J.P., Retired Dep. Surgeon-General, Bengal
Army, Montpellier Hall, Brighton. *Councillor.*
1877. EWART, WILLIAM, M.D., 33, Curzon-street, Mayfair, W. c.

1889. FAIRBANK, FREDERICK ROYSTON, M.D., 59, Warrior-square, St. Leonards-on-Sea.
1884. FARDON, EDWARD ASHBY, M.R.C.S., Middlesex Hospital, W.
1873. FAYRER, Sir JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., 53, Wimpole-street, W. p, vp, ll, sm, o, c.
1884. FENTON, FREDERICK ENOS, F.R.C.S., Langstone, Ealing, W.
1888. FENWICK, BEDFORD, M.D., 20, Upper Wimpole-street, W.
1885. FENWICK, EDWIN HURRY, F.R.C.S., 5, Old Burlington-street, W.
1887. FERRIER, DAVID, M.D. Edin., F.R.S., 34, Cavendish-square, W.
1878. FIELD, GEORGE, F.R.C.S., 34, Wimpole-street, W. *Councillor*.
1883. FINLAY, DAVID WHITE, M.D., The University, Aberdeen. c 2.
1876. FISHER, FREDERIC RICHARD, F.R.C.S., 17, Wimpole-street, W.
1884. FLINT, ARTHUR, M.D., Westgate Lodge, Westgate-on-Sea.
1878. *FONMARTIN, HENRY DE, M.D., 1, Anchor-gate-terrace, Portsea, Hants.
1884. FOTHERBY, HENRY ARTHUR, 20, Warwick-street, Kensington, W.
1879. FOWLER, JAMES KINGSTON, M.D., 35, Clarges-street, Mayfair, W. s 2, c.
1873. FOX, ARTHUR EDWARD WELLINGTON, M.B., C.M., 16, Gay-street, Bath. c.
1887. FOX, FORTESCUE, M.B., Strathpeffer Spa, Ross-shire.
1871. FOX, FRANCIS, M.R.C.S., 68, Wimpole-street, W. c 3.
1885. FOX, R. HINGSTON, M.D., 23, Finsbury-square, E.C.
1879. FOX, THOMAS COLCOTT, M.B., 14, Harley-street, W. s 2. *Councillor*.
1887. FRAZER, ROBERT FAIR, 185, Lavender-hill, New Wandsworth, S.W.
1868. FREER, ALFRED, J.P., Stourbridge, Worcestershire.
1886. FRITH, BAPTIST GAMBLE, M.B., 29, Cornwallis-gardens, Hastings.
1884. FROST, WILLIAM ADAMS, F.R.C.S., 17, Queen Anne-street, W. c 2.
1883. GABBETT, HENRY SINGER, M.D., 8, Chiswick-place, Eastbourne.
1862. GANT, FREDERICK JAMES, F.R.C.S., 16, Connaught-square, W. p, vp 2, ll, o, c 3.
1847. *GARROD, Sir ALFRED BARING, M.D., F.R.S., 10, Harley-street, W. p, vp 2, ll, o, c 9.
1887. GARROD, ARCHIBALD EDWARD, M.D., 9, Chandos-street, Cavendish-square, W.
1891. GASTER, AUGHEL, M.D., 34, Warwick-road, Maida-vale, W.
1887. GAY, JOHN, 119, Upper Richmond-road, Putney, S.W.
1879. GIBBES, HENEAGE, M.D., The University, Michigan, U.S.A.
1856. GIBBON, SEPTIMUS, M.B., 39, Oxford-terrace, W.
1882. GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan-place, S.W.
1881. GIFFARD, DOUGLAS W., M.R.C.S., 5, Pavilion-parade, Brighton.
1867. GILL, WILLIAM, M.R.C.S., 11, Russell-square, W.C. c.
1869. GODSON, CLEMENT, M.D., 9, Grosvenor-street, W. vp, c 3, s 2, sm.

1873. GOODSALL, DAVID HENRY, F.R.C.S., 17, Devonshire-place, Portland-place, W. c, s 2, SM, CFC 6. *Vice-President; Chairman, House and Finance Committee.*
1892. GORDON, ROBERT JOHN, M.B., 42, Grove-road, Regent's-park, N.W.
1880. GOUDE, HERBERT, M.D., Smallpox Hospital, Highgate-hill, N.
1878. *GOULD, ALFRED PEARCE, M.S., 10, Queen Anne-street, W. s 2, c 3.
1876. GOWERS, WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne-street, W.
VP, C, SM, LL.
1874. GOWLLAND, PETER YEAMES, F.R.C.S., 34, Finsbury-square, E.C.
1887. GRANT, JAMES EDWARD RONEY, 2, Charing Cross-chambers, Duke-street, Adelphi, W.C.
1881. GREEN, THOMAS HENRY, M.D., 74, Wimpole-street, W. c 2. VP.
1868. GREGSON, GEORGE, M.R.C.S., 63, Harley-street.
1886. GREVES, EDWIN HYLIA, M.D., Rodney House, Bournemouth.
1873. GRIEVE, ROBERT, M.D., British Guiana.
1884. GRIFFITH, DAVID CHARLES BALLINGER, M.R.C.P. Edin., 3, Lansdowne-place, Brighton.
1875. GRIFFITH, G. DE GORREQUER, M.R.C.S., 34, St. George's-square, S.W.
1885. GRIFFITHS, CHARLES THOMAS, L.R.C.P., 206, Lozells-road, Birmingham.
1884. GRIFFITHS, HERBERT TYRRELL, M.B., 5, Kensington-square, W.
1880. GRISTOCK, WILLIAM, M.D. Lond., 6, Finchley-road, N.W.
1891. GUTHRIE, LEONARD G., M.B., 24, Upper George-street, Bryanston-square, W.
1886. HABERSHON, S. HERBERT, M.D., 70, Brook-street, Grosvenor-square, W.
1884. HADDEN, WALTER BAUGH, M.D., 21, Welbeck-street, Cavendish-square, W. c 2.
1891. HADLEY, WILFRED J., M.B., 16, Wimpole-street, W.
1887. HAIG, ALEXANDER, M.B., 7, Brook-street, W.
1884. HAIRSINE, HUDSON, Roose House, Upper Tooting, S.W.
1881. HALL, CHARLES ROSS, M.R.C.S., Hatfield, Herts.
1874. *HALL, FRANCIS DE HAVILLAND, M.D., 47, Wimpole-street, W. c 4, s 2, SM. *Vice-President.*
1885. HALPIN, RICHARD FREDERICH BESTALL, Arklow, co. Wicklow, Ireland.
1881. HAMES, GEORGE HENRY, F.R.C.S., 29, Hertford-street, Mayfair, W.
1879. HAMILTON, SETON GUTHRIE, Surgeon-Captain, 1st Life Guards.
1891. HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish-square, W.
1887. HANDFORD, HENRY, M.D. Edin., 14, Regent-street, Nottingham.
1850. *HARE, CHARLES JOHN, M.D., *Honorary Fellow (q. v.).*
1888. HARE, HOBART AMORY, 117, South Twenty-second-street, Philadelphia.
FM. 1888.

1891. HAROLD, JOHN PATRICK, M.R.C.S., 72, Wimpole-street, W.
 1882. HARPER, GERALD SAMUEL, M.B., 40, Curzon-street, Mayfair, W.
 1871. HARRIS, CHARLES JAMES, 4, Kilburn Priory, N.W.
 1873. HARRIS, WILLIAM JOHN, Church House, Heene, Worthing.
 1871. HARRISON, REGINALD, F.R.C.S., 6, Lower Berkeley-street, Portman-square. VP 2, LL, C 1.
 1883. *HARTRIDGE, GUSTAVUS, F.R.C.S., 65, Green-street, Grosvenor-square, W.
 1864. HARVEY, JOHN ALEXANDER, 35, Princes-square, Bayswater, W.
 1882. HARVEY, JOHN STEPHENSON SELWYN, M.D., 1, Astwood-road, Cromwell-road, S.W.
 1882. HASLAM, WILLIAM FREDERICK, F.R.C.S., 33, Paradise-street, Birmingham. C 2. *Councillor*.
 1852. *HAWARD, EDWIN, M.D., 34A, Gloucester-place, W.
 1883. HAWKEN, CHARLES ST. AUBYN, 20, North-terrace, Wandsworth, S.W.
 1889. HAWKINS, FRANCIS HENRY, M.B., 59, Wimpole-street, W.
 1890. HEBB, FREDERICK THEODORE, M.R.C.S., 7, Milner-terrace, Cadogan-square, S.W.
 1884. HENSMAN, ARTHUR, F.R.C.S., 31, Harley-street, W.
 1891. HENSMAN, FRANK, M.R.C.S., Surgeon-Major, 1st Life Guards.
 1883. HERMAN, GEORGE ERNEST, M.B., 20, Harley-street, W.
 1879. HERON, GEORGE ALLAN, M.D., 57, Harley-street, Cavendish-square, W. C. *Councillor*.
 1886. HERRINGHAM, WILMOT PARKER, M.B., 13, Upper Wimpole-street, W.
 1883. HERSHELL, GEORGE A., M.D., 5, West-street, Finsbury-circus, E.C.
 1883. HEWITT, FREDERICK WILLIAM, M.D., 10, George-street, Hanover-square, W.
 1876. HEYCOCK, FRANCIS RAWORTH, C.M., 26, Upper Wimpole-street, W.
 1872. HICKS, JOHN BRAXTON, M.D., F.R.S., 34, George-street, Hanover-square. C.
 1892. HILL, WILLIAM, M.D., 24, Wimpole-street, W.
 1873. HOBSON, WILLIAM HENRY, M.R.C.S., Great Berkhamstead, Herts.
 1879. HOGG, ARTHUR JOHN, M.R.C.S., Leslie Lodge, Haven-green, Ealing, W.
 1884. HOLLAND, CHARLES EDWARD, M.B., 44, Warwick-road, Maida-vale, W.
 1888. HOLM, JOHN, F.R.C.S. Edin., 13, Stratford-place, W.
 1868. HOLMAN, CONSTANTINE, M.D., The Baron's, Reigate. C 4.
 1881. HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green-street, W. C. *Councillor*.
 1879. HOOKHAM, PAUL (address uncommunicated).
 1875. HOPE, WILLIAM, M.D., 56, Curzon-street, Mayfair, W.
 1883. *HOVELL, T. MARK, F.R.C.S. Edin., 105, Harley-street, W.
 1886. HUDDART, CUTHBERT HENRY COOKE, M.B., Shoyswell Manor, Etchingam, Sussex.
 1885. HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, F.R.C.S., 6, Chandos-street, W.

1890. HUGHES, EDGAR, F.R.C.S., 10, Old Cavendish-street, W.
 1864. HUME, FREDERICK HENRY, M.D., 53, Devonshire-street, Islington, N.
 1884. HUNTER, SIR WILLIAM GUYER G., M.D., 21, Norfolk-crescent, Hyde Park, W.
 1889. HUNTER, WILLIAM, M.D., 61, Wimpole-street, W.
 1881. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish-square. LL C 4, o. *President.*
 1875. HUTCHINSON, SAMUEL JOHN, M.R.C.S., 64, Brook-street, W.
 1889. I'ANSON, WILLIAM ANDREW, Westgate Hill House, Newcastle-on-Tyne.
 1891. ISAAC, GEORGE WASHINGTON, M.B., 7, Mornington-crescent, N.W.
 1884. *JACKSON, FREDERICK WILLIAM, M.D., Yorkgate House, Broadstairs.
 1885. JACKSON, JAMES, M.R.C.S., 15, Huntingdon-street, Barnsbury, N.
 1868. JACKSON, JOHN HUGHLINGS, M.D., F.R.S., 3, Manchester-square, W. P, VP, O, C 5.
 1853. JACKSON, ROBERT, M.D., 53, Notting-hill-square, W.
 1874. JAGIELSKI, VICTOR APOLLINARIS, M.D., 54, York-terrace, Regent's Park, N.W.
 1882. JAMES, JOSEPH BRINDLEY, M.R.C.S., 47, Jamaica-road, Bermondsey, S.E.
 1887. JAMISON, ARTHUR ANDREW, M.D., 18, Lowndes-street, Belgrave-square, S.W.
 1884. JENNINGS, CHARLES EGERTON, F.R.C.S., 48, Seymour-street, W.
 1886. JERVIS, ARTHUR, M.R.C.S., Seamen's Hospital, Greenwich, S.E.
 1883. JESSETT, FREDERICK BOWREMAN, F.R.C.S., 1, Buckingham Palace-mansions, S.W.
 1883. JESSOP, WALTER HAMILTON, F.R.C.S., 73, Harley-street, W.
 1886. JOHNSTON, JAMES, M.D., 11, Chester-place, Hyde Park-square, W.
 1888. JONES, ARTHUR HENRY, M.D., 45, Sheep-street, Northampton.
 1890. JONES, H. MACNAUGHTON, M.D., 141, Harley-street, Cavendish-square, W.
 1888. JONES, JOHN TALFOURD, M.B., Rosebank, South-terrace, Eastbourne, Sussex.
 1892. *JONES, ROBERT, F.R.C.S., 11, Nelson-street, Liverpool.
 1881. JONES, THOMAS WILLIAM CARMALT, F.R.C.S. Edin., 6, Westbourne-street, Hyde Park, W.
 1877. JULER, HENRY EDWARD, F.R.C.S., 77, Wimpole-street, W.
 1889. KAUFFMANN, OTTO JACKSON, M.D., Queen's Hospital, Birmingham.
 1874. KAVANAGH, PATRICK, M.D., 81, Marine-parade, Brighton.
 1891. KEEGAN, DENIS FRANCIS, M.D., Surgeon-Major, The Residency, Indore, Central India.
 1884. KEETLEY, CHARLES BELL, F.R.C.S., 56, Grosvenor-street, W. c 2. *Councillor.*

1847. *KELLOCK, WILLIAM BERRY, M.D., 84, Stamford-hill, N.
 1890. KELLY, AUGUSTIN BERNARD, M.R.C.S., 82, Park-street, Grosvenor-square, W.
 1891. KELSON, WILLIAM HENRY, M.D., 46, Watling-street, E.C.
 1883. KEMP, JOHN ROBERT, M.R.C.S., 101, Jermyn-street, S.W.
 1890. KER, HUGH RICHARD, F.R.C.S. Edin., Devonshire Cottage, Balham Hill, S.W.
 1884. KERR, NORMAN, M.D., 42, Grove-road, N.W.
 1881. KESER, JEAN SAMUEL, M.D., 11, Harley-street, W. c 2. *Honorary Secretary for Foreign Correspondence.*
 1876. KEY, AUGUSTUS COOPER, M.R.C.P. Edin., 30, Wilton-place, S.W. *Councillor.*
 1886. KIDD, PERCY, M.D., 60, Brook-street, W. c. *Councillor.*
 1889. KIRKHAM, FREDERICK WILLIAM, Downham Market, Norfolk.
 1883. KNAPTON, GEORGE, Cliveden House, 4, Cliveden-place, Eaton-square, S.W.
 1875. KNOX, JOHN, M.D., Bethnal Green Infirmary, E.

 1889. LAKE, RICHARD, F.R.C.S., Exeter House, Barnes, S.W.
 1868. LAKE, WILLIAM CHARLES, M.D., Teignmouth, Devon.
 1881. LANGTON, JOHN, F.R.C.S., 62, Harley-street, W. c 2.
 1882. LARKIN F. COLET, M.B., Kingsbridge House, Avenue-road, East Cliff, Ramsgate.
 1890. LAW, EDWARD, M.D. Edin., 35, Harley-street, W.
 1890. LAWRIE, EDWARD, M.B. Edin., Surgeon-Major, Bengal Army, The Residency, Hyderabad.
 1858. LAWSON, GEORGE, F.R.C.S., 12, Harley-street, W. vp 2, c 3.
 1891. LAZARUS-BARLOW, WALTER SYDNEY, M.B., 16, Bryanston-street, Portman-square, W.
 1887. LEGGATT, CHARLES ASHLEY SCOTT, M.D., 2, Walton-place, S.W.
 1858. LEMON, OLIVER, M.R.C.S., 12, The Grange, Highbury, N.
 1886. LEWERS, ARTHUR HAMILTON NICHOLSON, M.D. Lond., 60, Wimpole-street, W.
 1867. LICHTENBERG, GEORGE, M.D., 47, Finsbury-square. c 2.
 1878. LISTER, Sir JOSEPH, Bart., D.C.L., LL.D., F.R.C.S., F.R.S., 12, Park-crescent, Portland-place. o.
 1890. LITHGOW, ROBERT ALEXANDER DOUGLAS, M.D., 27A, Lowndes-street, S.W.
 1889. LITTLE, ERNEST MUIRHEAD, F.R.C.S., 40, Seymour-street, Portman-square, W.
 1889. LITTLE, FLETCHER, M.D., 60, Welbeck-street.
 1887. LLOYD, ROBERT HODGENS, M.D., Brook House, Kennington-road, S.E.
 1886. LLOYD, SAMUEL, M.D., 4, High-street, Bloomsbury, W.C.
 1878. LOCKWOOD, CHARLES BARRETT, F.R.C.S., 19, Upper Berkeley-street, W. c.

1873. LOE, JAMES SCARBOROUGH, 26, Woodhouse-lane, Leeds.
1881. LORIMER, G., M.D., Buxton, Derbyshire.
1868. LOWE, JOHN, M.D., J.P., 4, Gloucester-place, Portman-square, W.
c 3.
1868. *LUND, EDWARD, F.R.C.S., 22, St. John's-street, Manchester. o, c 3.
1889. LUNN, HENRY SIMPSON, M.D., 5, Endsleigh-gardens, N.W.
1889. LUSH, PERCY, M.B., 8, Fitzjohn's-avenue, Hampstead, N.W.
1884. MACBRYAN, HENRY CRAWFORD, Kingsdown House, Box, Wilts.
1871. MACCORMAC, SIR WILLIAM, F.R.C.S., 13, Harley-street, W. p, vp,
§ 2, c 4, o.
1882. MACKELLAR, ALEXANDER OBERLIN, F.R.C.S., 79, Wimpole-street, W.
1880. MACKENZIE, STEPHEN, M.D., 18, Cavendish-square, W. vp 2, c 2,
ll. *Councillor*.
1881. MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan-place, S.W. c 3.
1861. MACLAREN, ALEXANDER CONNELL, 60, Harley-street, W.
1891. MACLEAN, ALLAN, Harpenden Hall, Herts.
1887. MACREADY, JONATHAN FOSTER CHRISTIAN HORACE, F.R.C.S., 51,
Queen Anne-street, W.
1883. MADDICK, EDMUND DISTIN, F.R.C.S. Edin., 2, Chandos-street, Caven-
dish-square, W.
1885. MAGUIRE, ROBERT, M.D., 4, Seymour-street, W. c 2.
1878. MAIR, ROBERT SLATER, M.D., 28, Ledbury-road, Bayswater, W.
1890. MALCOLM, JOHN DAVID, F.R.C.S. Edin., 13, Portman-street, Portman-
square, W.
1887. MANTLE, ALFRED, M.D., 12, Park-road, Halifax.
1888. MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish-square, W.
1891. MARSH, HOWARD, F.R.C.S., 30, Bruton-street, W.
1892. MARSHALL, ARTHUR LUMSDEN, M.B., 56, Rectory-road, N.
1873. MARSHALL, EDWARD, M.R.C.S., Mitcham, Surrey.
1869. MARSHALL, WILLIAM, M.D., Torrieburn, Barnes, S.W.
1864. MARSHALL, WILLIAM GURSLAVE, F.R.C.S., 72, Bromfelde-road, Clap-
ham, S.W.
1889. MARTIN, JOHN MICHAEL HARDING, M.D., Arnheim, Blackburn, Lan-
cashire.
1890. MARTIN, SIDNEY, M.D., 10, Mansfield-street, W.
1884. MATHESON, FARQUHAR, M.B., 11, Soho-square, W.
1891. MAUDE, ARTHUR, M.R.C.S., Winterton House, Westerham, Kent.
1892. MAUNSELL, H. WIDENHAM, M.D., 37, Stanhope-gardens, Queen's-gate,
S.W.
1891. MAY, WILLIAM PAGE, M.D., National Hospital for Paralysis, Queen-
square, W.C.
1885. McCONNEL, HENRY WILSON, M.B., Litchdon, Barnstaple, Devon.
1885. McGEAGH, THOMAS EDWIN FOSTER, M.D., 23, New Cavendish-street,
W.

1873. ^TMcHARDY, MALCOLM MACDONALD, F.R.C.S. Edin., 5, Savile-row, W.
 1884. MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester-square, W.
 1864. MIDDLEMIST, ROBERT PERCY, M.R.C.S., 6, Devonport-street, Hyde Park, W. c 4.
 1882. MILLS, JOSEPH, M.R.C.S., 28, Queen Anne-street, Cavendish-square, W.
 1883. MONEY, ANGEL, M.D., 24, Harley-street, W. c.
 1883. MOORE, THOMAS, F.R.C.S., 6, Lee-terrace, Blackheath, S.E.
 1883. MORGAN, JOHN HAMMOND, F.R.C.S., 68, Grosvenor-street, W. s 2, c.
 1871. MORLEY, ALEXANDER, 42, Albemarle-street, W.
 1881. MORRIS, HENRY, F.R.C.S., 8, Cavendish-square, W. c.
 1878. MORRIS, MALCOLM ALEXANDER, F.R.C.S. Edin., 8, Harley-street, W. c 2.
 1882. MORTON, ANDREW STANFORD, F.R.C.S., 26, Weymouth-street, Portland-place, W.
 1884. *MOULLIN, CHARLES WILLIAM MANSELL, F.R.C.S., 69, Wimpole-street, W.
 1878. MUMFORD, WILLIAM LUGAR, M.D., 12, Suffolk-street, Pall-mall, S.W.
 1836. MURPHY, JAMES, M.D., Holly House, Sunderland.
 1884. MURRAY, FRED., M.B., Durbanville, Cape Colony, South Africa.
 1890. MURRAY, GEORGE, M.R.C.S., 34, Wimpole-street, Cavendish-square, W.
 1886. MURRAY, HUBERT MONTAGUE, M.D., 27, Savile-row, W.
 1883. MURRAY, JAMES, M.D., 21, Weymouth-street, W.
 1879. MURRELL, WILLIAM, M.D., 17, Welbeck-street, W.
 1885. MYERS, ARTHUR THOMAS, M.D., 2, Manchester-square, W.
1877. NESBITT, DAWSON, M.D., 1, Norfolk-square, Hyde Park, W.
 1876. NEWHAM, JAMES, 80, Gloucester-place, W.
 1889. *NIAS, J. BALDWIN, M.B., 40, Brook-street, Grosvenor-square, W.
 1880. NIX, EDWARD JAMES, M.D., 11, Weymouth-street, W.
1887. OAKLEY, ADAM ROBERT HAMILTON, L.R.C.P., Treath, Hornchurch, Essex.
 1885. OGILVIE, LESLIE, M.B., 46, Welbeck-street, W.
 1884. OGLE, CHARLES JOHN, 1, Cavendish-place, W.
 1884. OLIVER, GEORGE, M.D., West End Park, Harrogate.
 1875. ORD, WILLIAM MILLER, M.D., 37, Upper Brook-street, W. p, c 4.
 1892. ORD, WILLIAM WALLIS, M.D., 32, Harley-street, W.
 1887. ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole-street, W. *Councillor.*
 1889. ORTON, GEORGE HUNT, M.B., 1A, Campden Hill-road, Kensington, W.
 1884. ORWIN, ARTHUR WIGELSWORTH, M.D., 15, Weymouth-street, Portland-place, W.
 1880. OSWALD, JAMES WADDELL JEFFREYS, M.D., 245, Kennington-road, S.E.
 1883. OWEN, CHARLES J. RAYLEY, 14, Devonshire-terrace, W.

1878. *OWEN, EDMUND, F.R.C.S., 64, Great Cumberland-place, W. VP 2, c 3,
s 2, SM, LL. *Trustee*.
1881. OWEN, ISAMBARD, M.D., 40, Curzon-street, Mayfair, W. s 2, c 4.
1886. PAGET, STEPHEN, F.R.C.S., 57, Wimpole-street, W. c. *Councillor*.
1880. PALMER, FREDERICK STEPHEN, M.D., Compton Lodge, East Sheen, S.W.
1882. PALMER, WILLIAM PITT, M.B., 17, Belgrave-terrace, Torquay.
1877. *PARAMORE, RICHARD, M.D., 2, Gordon-square, W.C.
1867. PARKINSON, GEORGE, 50, Brook-street, Grosvenor-square, W.
1880. PARKINSON, GEORGE WILLIAM, 1, Bentinck-street, Cavendish-square,
W.
1881. PARROTT, EDWARD JOHN, M.R.C.S., Hayes, Uxbridge, Middlesex.
1871. PARSONS, FRANCIS HENRY, M.D., "The Hurst," West Worthing.
1885. PASTEUR, WILLIAM, M.D., 4, Chandos-street, Cavendish-square, W.
Honorary Secretary.
1872. PATTEN, CHARLES ARTHUR, M.R.C.S., Marpool House, Ealing, W.
1891. PATTERSON, CHARLES SUMNER, M.B., 347, City-road, E.C.
1890. PATTISON, EDWARD SETON, M.R.C.S., Granville House, Fulham-park,
S.W.
1861. PAUL, JOHN HAYBALL, M.D., Camberwell House, Camberwell, S.E. c 6.
1854. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor-street, W.
VP, LL, C.
1881. *PEACEY, WILLIAM, M.B., 11, Breakspears-road, Brockley, S.E.
1883. PECK, EDWARD GEORGE, M.A., Queensbury, Bradford, Yorks.
1871. PEDLER, GEORGE HENRY, M.R.C.S., 6, Trevor-terrace, Knightsbridge,
S.W.
1883. PERIGAL, ARTHUR, M.D., New Barnet, Herts.
1876. PHILLIPS, CHARLES DOUGLAS FERGUSON, M.D., F.R.S.E., 10, Henri-
etta-street, Cavendish-square, W. c 3.
1873. PHILLIPS, GEORGE RICHARD TURNER, M.R.C.S., 24, Leinster-square,
Hyde Park, W. c 2.
1885. PHILLIPS, JOHN, M.D., 71, Grosvenor-street, W.
1883. PHILLIPS, SIDNEY PHILIP, M.D. Lond., 62, Upper Berkeley-street,
Portman-square, W.
1878. PHILLIPS, SUTHERLAND REES, M.D., St. Ann's Heath, Virginia Water,
Berks.
1883. PICK, THOMAS PICKERING, F.R.C.S., 18, Portman-street, W. c 2.
1884. PIESSE, C. H., M.R.C.S., 2, New Bond-street, W.
1883. PITTS, BERNARD, F.R.C.S., 31, Harley-street, Cavendish-square. c 5, s 2.
1890. POPE, HARRY CAMPBELL, M.D. Lond., 280, Goldhawk-road, Shep-
herd's Bush, W.
1873. PORT, HEINRICH, M.D., 48, Finsbury-square, E.C.
1850. *POTTS, WILLIAM, F.R.C.S., 2, Albert-terrace, Regent's Park, N.W. c 3.
1871. POWELL, RICHARD DOUGLAS, M.D., 62, Wimpole-street, W. P, VP,
c 4, O. *Councillor*.

XXXVIII

1891. POWELL, WILLIAM WYNDHAM, M.R.C.S., 9, Cliveden-place, Eaton-square, S.W.
1891. PRESTON, THEODORE JULIAN, M.R.C.S., Staff Surgeon, Royal Navy, 7, Caroline-place, Mecklenburgh-square, W.C.
1891. *PRICKETT, MARMADUKE, M.D., 12, Devonport-street, Gloucester-square, W.
1885. PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour-street, W.
1889. PRITCHARD, OWEN, M.D., 37, Southwick-street, Hyde Park, W.
1873. PURCELL, FERDINAND ALBERT, M.D., 7, Manchester-square, W.
1870. QUAIN, Sir RICHARD, Bart., M.D., F.R.S., 67, Harley-street, W. vp, c 3.
1883. RALFE, CHARLES HENRY, M.D., 26, Queen Anne-street, W.
1861. RAMSKILL, JABEZ SPENCE, M.D., 5, St. Helen's-place, E.C.
1881. RANKING, JOHN EBENEZER, M.D., 18, Mount Ephraim-road, Tunbridge Wells.
1859. *RAYNER, JOHN, M.R.C.P. Edin., Swaledale House, Highbury-quadrant, N.
1890. RAYNER, WILLIAM, M.R.C.S., 4, Dorset-square, N.W.
1850. *READ, REGINALD, F.R.C.P. Edin., 15 Windsor-road, Denmark-hill, S.E.
1879. REEVES, HENRY ALBERT, F.R.C.S. Edin., 7, Grosvenor-street, W.
1890. REID, JOHN, M.B., Clanmurray, Dromore, co. Down.
1882. REID, THOMAS WHITEHEAD, F.R.C.P. Edin., 34, St. George's-place, Canterbury.
1887. REMFRY, LEONARD, M.D., 4, Harley-street, W.
1872. REYNOLDS, JOHN RUSSELL, M.D., F.R.S., 38, Grosvenor-street, W. c 3.
1872. RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland-road, Ealing, W. c. 2.
1850. *RICHARDSON, BENJAMIN WARD, M.D., LL.D., F.R.S., 25, Manchester-square, W. p, vp, LL, c 5, o, FM 1854.
1830. *ROBARTS, HENRY PRATT, F.R.C.S., 31, Great Coram-street, W.C. vp 2, s 9, c 10, FM 1844, SM.
1891. ROBB, JAMES TAYLOR, M.D., 33, Lowndes-street, Belgrave-square, W.
1868. *ROBERTS, DAVID LLOYD, M.D., F.R.S.E., 11, St. John's-street, Manchester.
1857. ROBERTS, DAVID WATKIN, M.D., 56, Manchester-street, W.
1885. ROBERTS, EDWARD COLERIDGE, M.R.C.S., Southgate, N.
1874. ROBERTS, FREDERICK THOMAS, M.D., 102, Harley-street, W.
1889. ROBERTS, Sir WILLIAM, M.D., F.R.S., 8, Manchester-square, W. c.
1873. ROBERTSON, WILLIAM HENRY, M.D., J.P., 6, The Square, Buxton, Derbyshire.
1884. ROBINSON, ARTHUR HENRY, M.D., The Infirmary, Bancroft-road, N.E.
1847. *ROGERS, WILLIAM RICHARD, M.D., 56, Berners-street, W. vp, c 6.
1890. ROOT, ARTHUR GUERNSEY, M.D., Albany, New York, U.S.A.

1886. ROSE, ROBERT DUNCAN, F.R.C.S., St. Leonard's-place, York.
1874. ROSE, WILLIAM, F.R.C.S., 17, Harley-street. c 2, LL. *Vice-President*.
1883. *ROSS, DANIEL MCCLURE, F.R.C.S., 54, Upper Berkeley-street, W.
1888. ROTH, BERNARD, F.R.C.S., 29, Queen Anne-street, W.
1876. ROUTH, ALFRED CURTIS, 33, Marina, St. Leonards-on-Sea.
1881. ROUTH, AMAND, M.D., 14A, Manchester-square, W. c 2.
1848. *ROUTH, CHARLES HENRY FELIX, M.D., 52, Montagu-square, W. p,
VP 2, O, LL, s 4, c 6, SM. *Trustee*.
1891. RUFFER, MARC ARMAND, M.D., 19, Iddesleigh-mansions, Victoria-street,
S.W.
1887. RUSHWORTH, FRANK, M.D., "Langdale," Goldhurst-terrace, South
Hampstead, N.W.
1889. RUSSELL, JAMES SAMUEL RISIEN, M.B., 94, Wimpole-street, W.
1886. RUTHERFOORD, HENRY TROTTER, M.D., 46, Queen Anne-street, W.
1879. RYLEY, JAMES BERESFORD, M.D., 1, Bentinck-street, W.
1887. SAINSBURY, HARRINGTON, M.D., 63, Welbeck-street, W.
1884. SALTER, THOMAS KNIGHT, M.R.C.S.
1863. *SANSOM, ARTHUR ERNEST, M.D., 84, Harley-street, W. VP, s 2, c 5,
SM, §, LL, O.
1886. SAVAGE, GEORGE HENRY, M.D., 3, Henrietta-street, W. c.
1886. SAVILL, THOMAS DIXON, M.D., Paddington Infirmary, W.
1873. SCOTT, WILLIAM, M.D., 34, New North-road, Huddersfield.
1873. SEDGWICK, JAMES, M.D., Boroughbridge, Yorkshire.
1868. SEDGWICK, LEONARD WILLIAM, M.D., 2, Gloucester-terrace, Hyde Park,
W. VP 2, c 4, § 3.
1883. SEMON, FELIX, M.D., 39, Wimpole-street, W. §, c.
1887. SERVAIS, LEOPOLD, M.D., Antwerp, Belgium.
1876. SEWELL, CHARLES BRODIE, M.D., 21, Cavendish-square W. c.
1889. SHAW, GEORGE, M.B., 46, Western-road, Hove.
1884. SHAW, JOHN, M.D., Burlington House, Willoughby-road, Hampstead,
N.W.
1886. SHEILD, ARTHUR MARMADUKE, F.R.C.S., 20, Stratford-place, Oxford-
street, W. c. *Honorary Secretary*.
1890. SHEPPARD, WILLIAM JOHN, M.D., Laurel House, Putney, S.W.
1881. SHIPTON, ARTHUR, F.R.C.S. Edin., Buxton, Derbyshire.
1878. SHIPTON, WILLIAM PARKER, M.R.C.S., J.P., Buxton, Derbyshire.
1885. SHOEMAKER, JOHN V., M.D., 1031, Walnut-street, Philadelphia, U.S.A.
1890. SILK, JOHN FREDERICK WILLIAM, M.D., 29, Weymouth-street, W.
1890. SIMON, ROBERT M., M.D., 27, Newhall-street, Birmingham.
1884. SIMPSON, JAMES HERBERT, M.D., The Crescent, Rugby, Warwickshire.
1884. SINCLAIR, JOHN, M.R.C.P., General Post Office, St. Martin's-le-Grand,
E.C.
1891. SISLEY, RICHARD, M.D., 11, York-street, Portman-square, W.
1883. *SKERRITT, EDWARD MARKHAM, M.D., Richmond Hill, Clifton.

1886. SLATER, CHARLES, M.B., 16, Northwick-terrace, St. John's Wood, N.W.
1862. SLIGHT, GEORGE, M.D., 14, Old Burlington-street, W. c 2.
1889. SMALE, MORTON, M.R.C.S., 22A, Cavendish-square, W.
1845. *SMILES, WILLIAM, M.D., St. Martha's Lodge, Guildford. vp 2, s 4, c 9, SM.
1887. SMITH, FREDERICK JOHN, M.D., 4, Christopher-street, Finsbury-square, E.C.
1848. *SMITH, HENRY, F.R.C.S., 7, Wimpole-street, W. p, vp, LL, G, c 3.
1882. SMITH, HERBERT, Urmson, Oudtshorne, Cape of Good Hope, South Africa.
1873. SMITH, HEYWOOD, M.D., 18, Harley-street, W. c 3.
1880. SMITH, NOBLE, F.R.C.S. Edin., 24, Queen Anne-street, W.
1891. SMITH, SOLOMON CHARLES, M.D., Savile-place, Halifax, Yorks.
1877. SMITH, SYDNEY LLOYD, M.R.C.S., 25, Argyle-square, King's Cross, W.C.
1882. SMITH, THOMAS FREDERICK HUGH, F.R.C.S., Farningham, Kent.
1873. *SMITH, THOMAS GILBART, M.D., 68, Harley-street, W. vp 2, s 2, SM, c 4. *Trustee.*
1872. SMITH, WALTER, M.R.C.P. Edin., 2, Stanhope-terrace, Gloucester-gate, Regent's Park, N.W.
1874. SMYTH, WILLIAM WOODS, Maidstone.
1888. SPENCER, JOHN, Lyons-terrace, Hetton, Durham.
1869. SPENDER, JOHN KENT, M.D., 17, Circus, Bath. fm 1874.
1887. SPICER, SCANES, M.D., 28, Welbeck-street, W.
1883. SPITTA, EDMUND JOHNSON, M.R.C.S., Ivy House, Clapham Common, S.W.
1864. SQUIRE, ALEXANDER JOHN BALMANNO, M.B., 24, Weymouth-street, Portland-place.
1881. STARTIN, JAMES, M.R.C.S., 15, Harley-street, W.
1892. STAVELEY, WILLIAM H. C., F.R.C.S., 108, Ebury-street, S.W.
1884. STEPHENS, WILLIAM JOHN, 41, Grand-parade, Brighton.
1882. STEWART, JAMES, F.R.C.P. Edin., Dunmurry, Sneyd-park, near Clifton.
1891. STEWART, WILLIAM EDWARD, F.R.C.S. Edin., 16, Harley-street, W.
1883. STEWART, WILLIAM ROBERT HENRY, F.R.C.S. Edin., 41, Devonshire-street, Portland-place, W.
1884. STIVEN, EDWARD WINNAN FLEMING, M.D., Lincoln House, Harrow, Middlesex.
1885. STIVENS, B. H. LYNE, M.D., 11, Kensington Gardens-square, W.
1848. *STOCKER, JOHN SHERWOOD, M.D., 2, Montagu-square, W. c 10, s 2.
1884. STOKER, GEORGE, 14, Hertford-street, Mayfair, W.
1892. STONHAM, CHARLES, F.R.C.S., 4, Harley-street, W.
1877. STOWERS, JAMES HERBERT, M.D., 41, Finsbury-square, E.C.
1873. STRANGE, WILLIAM HEATH, M.D., 5, Grosvenor-street, W. c 3.
1881. STURGE, WILLIAM ALLEN, M.D., 9, Rue Longchamp, Nice. SM.
1889. SUMPTER, WALTER JOHN ERNELY, M.R.C.S., Sheringham, Norfolk.
1876. *SUTHERLAND HENRY, M.D., 6, Richmond-terrace, Whitehall, S.W.

1885. *SYERS, HENRY WALTER, M.D. Camb., 3, Devonshire-street, Portland-place, W.
1881. SYKES, EDWIN JOHN, M.B., York House, Tottenham.
1884. SYMONDS, HORATIO PERCY, F.R.C.S., 35, Beaumont-street, Oxford.
1885. TADLOCK, A. B., M.D., Knox Ville, Tennessee, U.S.A.
1864. TAIT, EDWARD WILMSHURST, 48, Highbury-park, N.
1879. *TAIT, LAWSON, F.R.C.S., 7, The Crescent, Birmingham.
1875. TAMPLIN, CHARLES HARRIS, "Lindfield," Crescent-road, Ramsgate.
1882. TAYLOR, SEYMOUR, M.D., 16, Seymour-street, Portman-square, W. c 2.
1884. TAYLOR, SYDNEY HAMILTON, M.D., 1, Park-avenue, Willesden-park, N.W.
1859. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish-square, W. vp, o, s 3, c 3, sm.
1855. THOMPSON, Sir HENRY, F.R.C.S., 35, Wimpole-street, W. vp., ll., c 4.
1873. THOMSON, JOHN ROBERTS, M.D., Monkchester, Bournemouth, Hants.
1884. THOMSON, WILLIAM SINCLAIR, M.D., 1, Palace-court, Notting-hill-gate, W.
1876. THORNTON, JOHN KNOWSLEY, M.C., 22, Portman-street, W. p, vp, c 3.
1867. THOROWGOOD, JOHN CHARLES, M.D., 61, Welbeck-street, W. ll, s 2, sm, c 3.
1856. THUDICHUM, JOHN LOUIS WILLIAM, M.D., 11, Pembroke-gardens Kensington, W. vp, ll, o, c.
1884. THURSFIELD, THOMAS WILLIAM, M.D., J.P., Selwood, Beauchamp-square, Leamington.
1876. TIBBITS, HERBERT, M.D., 68, Wimpole-street, W.
1867. TIMMS, GODWIN WILLIAM, M.D., 9, Wimpole-street, W.
1865. TRAVERS, WILLIAM, M.D., 2, Phillimore-gardens, Kensington.
Councillor.
1884. *TREVES, FREDERICK, F.R.C.S., 6, Wimpole-street, W. c 2.
1882. TUKE, CHARLES MOLESWORTH, Manor House, Chiswick.
1886. TUKE, THOMAS SEYMOUR, M.B. Oxon., Manor House, Chiswick.
1884. TURNER, GEORGE R., F.R.C.S., 49, Green-street, Grosvenor-square, W.
Councillor.
1890. TWEED, EDWARD REGINALD, M.D., 55, Upper Brook-street, W.
1883. TWEEDY, JOHN, F.R.C.S., 100, Harley-street, W.
1891. TYSON, WILLIAM JOSEPH, M.D. Durham, 10, Langhorne-gardens, Folkestone.
1887. *UNDERWOOD, EDWARD T., M.D., Fort Bombay, India.
1883. VENNING, EDGCOMBE, F.R.C.S., 30, Cadogan-place, S.W.
1874. VERLEY, REGINALD LOUIS, F.R.C.P. Edin., 28B, Devonshire-street, Portland-place, W.
1850. *WAGGETT, JOHN, M.D., Perivale, Bournemouth; and Union Club S.W.

1889. WAKEFIELD, THOMAS, M.B., 21, Beaumont-street, Marylebone, W.
1884. WAKLEY, THOMAS, 5, Queen's-gate, S.W.
1850. *WAKLEY, THOMAS HENRY, F.R.C.S., 5, Queen's-gate, S.W.
1880. WALSHAM, WILLIAM JOHNSON, F.R.C.S., 27, Weymouth-street, W. c.
1881. WARNER, FRANCIS, M.D., 5, Prince of Wales-terrace, W.
1883. WATERHOUSE, WILLIAM DAKIN, LL.D., 18, Woodchurch-road, West Hampstead, N.W.
1872. WATERS, JOHN, M.R.C.S., 41, Bloomsbury-square, W.C.
1868. WATKINS, CHARLES STUART, M.R.C.S., 16, King William-street, Strand, W.C.
1891. WATSON, W. SPENCER, F.R.C.S., 7, Henrietta-street, Cavendish-square, W.
1889. WAUGH, HENRY DUNN, M.D., 6, Sumner-place, Onslow-square, S.W.
1884. WEBB, F. ERNEST, M.R.C.S., 113, Maida-vale, W.
1889. WEBER, HERMANN, M.D., 10, Grosvenor-street, W.
1887. WEBSTER, HENRY WILLIAM, M.D., St. George's Infirmary, Fulham-road, S.W.
1838. *WELLS, JOHN ROBINSON, F.R.C.S., 4, Pierrepont-road, Acton, W. c 2.
1884. WEST, SAMUEL, M.D., 15, Wimpole-street, W. s 2, c 2, CFC. *Councillor.*
1889. WETHERED, FRANK JOSEPH, M.D., 34, Queen Anne-street, W.
1882. WHIPHAM, THOMAS T., M.D., 11, Grosvenor-street, W. sm, c.
1884. WHISTLER, WILLIAM MACNEILL, M.D., 17, Wimpole-street, W.
1889. WHITE, E. F., F.R.C.S., 7, Dealtry-road, Putney, S.W.
1868. WHITE, JOSEPH, F.R.C.S. Edin., Oxford-street, Nottingham.
1880. *WHITE, WILLIAM HENRY, M.D., 43, Weymouth-street, W. c 3.
1885. WHITE-COOPER, GEORGE OWEN, M.B., 5, Cranley-gardens, S.W.
1883. WHITEHEAD, WALTER, F.R.C.S. Edin., F.R.S.E., 499, Oxford-road, Manchester.
1885. WHITLA, WILLIAM, M.D., 8, College-square North, Belfast, Ireland.
1877. WHITMORE, WILLIAM TICKLE, 7, Arlington-street, Piccadilly, W.
1872. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook-street, Grosvenor-square, W. p, vp 2, LL, s 2, SM, o, L 3, c 9.
1876. WILLIAMS, HENRY WILLIAM, M.D., 7, Chapel-place, Cavendish-square, W.
1883. WILLIAMS, JOHN, M.D., 63, Brook-street. c 3.
1883. WILLIS, ARTHUR KEITH, M.A. Oxon., Gascony House, West End-lane, N.W.
1881. WILLS, CALEB SHERA, C.B., Brigade Surgeon, Lunecliffe, Lancaster.
1873. WILLS, THOMAS MUNNS, F.R.C.S.I., J.P., 44, Merton-road, Bootle, Liverpool.
1892. WILSON, CLAUDE, M.D., Belmont, Tunbridge Wells.
1884. WINSLOW, H. FORBES, M.D., 14, York-place, Portman-square, W.
1873. WINSLOW, LYTTELTON STEWART FORBES, D.C.L., M.B., 70, Wimpole-street, W. c.
1876. WOAKES, EDWARD, M.D., 78, Harley-street, W.
1882. WOLFENDEN, RICHARD NORRIS, M.D., 35, Harley-street, W.

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1886. WOOD, T. OUTTERSON, M.D., 40, Margaret-street, Cavendish-square, W.
c 3.
1873. WOODHOUSE, ROBERT HALL, M.R.C.S., 1, Hanover-square, W.
1889. WOOLFSON DE, LOUIS E. G., 26, St. John's-hill, Shrewsbury.
1891. WOOLLETT, CHARLES JEROME, F.R.C.S., 35, Telfourd-avenue, Streat-
ham-hill, S.W.
1886. WORDSWORTH, WILLIAM JOHN, 4, Tisbury-road, West Brighton.
1884. WYMAN, WILLIAM SANDERSON, M.D., Red Brae, Putney-hill, S.W.
c 2.
1891. YARR, MICHAEL THOMAS, Surgeon-Captain, Medical Staff, Junior Army
and Navy Club, St. James's-street, S.W.
1884. YEO, I. BURNEY, M.D., 44, Hertford-street, Mayfair, W.
1884. YOUNGER, EDWARD GEORGE, M.D., 19, Mecklenburgh-square, W.C.

NON-SUBSCRIBING FELLOWS.

1868. BATEMAN, Sir FREDERIC, M.D., J.P., Upper-street, Giles-street, Norwich.
1868. BEATTY, THOMAS CARLYLE, Seaham Harbour, Durham.
1872. BELL, JOHN HOUGHAM, M.D., Downside, Ventnor, Isle of Wight.
1868. BUCKLE, FLEETWOOD, M.D., Staff Surgeon R.N.
1868. CHILD, EDWIN, New Malden, Surrey.
1870. CLOUSTON, THOMAS SMITH, M.D., Royal Asylum, Morningside,
Edinburgh. fm 1870.
1868. FLETCHER, THOMAS BELL ELCOCK, M.D., J.P., 43, Clarendon-square,
Leamington.
1868. FOLKER, WILLIAM HENRY, F.R.C.S., Hanley, Staffordshire.
1869. FOSTER, Sir WALTER B., M.D., M.P., 14, Temple-row, Birmingham.
1863. FOX, CHARLES HENRY, M.D., Brislington House, near Bristol.
1868. FOX, JOHN MAKINSON, The Grove, Lymm, Cheshire.
1868. GAINE, CHARLES, 30, Gay-street, Bath.
1871. GLYNN, THOMAS ROBINSON, M.D., 62, Rodney-street, Liverpool.
1872. HARRIS, HENRY, LL.D., M.D., Redruth, Cornwall.
1868. KIRKMAN, WILLIAM PHILLIPS, M.D., Fearon-road, Hastings.
1868. KNAGGS, SAMUEL, Ebor Mount, Huddersfield.
1869. LEES, CHARLES ALEXANDER, M.D., Fleet Surgeon R.N.
1869. LIPSCOMB, JOHN THOMAS NICHOLSON, M.D., St. Albans, Herts.
1869. LUNN, WILLIAM JOSEPH, M.D., Hull.
1859. MARSHALL, JAMES, M.D., 6, Rubislaw-place, Aberdeen.

- 1869. MATHEWS, ROBERT, Bickley, Kent.
- 1871. MAURICE, OLIVER CALLEY, Reading.
- 1862. MAYBURY, AUGUSTUS KINGSTON, M.D., Holly Lodge, Richmond,
Surrey.
- 1868. MCINTYRE, JOHN, M.D., Odiham, Hants.
- 1868. NEVINS, JOHN BIRKBECK, M.D., 3, Abercromby-square, Liverpool.
- 1871. OGLE, WILLIAM, M.D., Derby.
- 1869. PEMBERTON, OLIVER, F.R.C.S., J.P., 65, Temple-row, Birmingham.
- 1869. PHILIPSON, GEORGE HARE, D.C.L., M.D., J.P., 7, Eldon-square,
Newcastle-on-Tyne.
- 1869. PRICE, WILLIAM PRESTON, M.D., 1, Ethelbert-crescent, Margate.
- 1869. PRIOR, CHARLES EDWARD, M.D., St. Peter's, Bedford.
- 1869. ROBERTS, BRANSBY, M.D., Badlesmere House, Eastbourne.
- 1871. SHETTLE, RICHARD CHARLES, M.D., 73, London-street, Reading.
- 1871. SLOMAN, SAMUEL GEORGE, Farnham.
- 1869. STEAR, HENRY, Saffron Walden, Essex.
- 1869. STEDMAN, JAMES REMINGTON, M.D., J.P., Guildford, Surrey.
- 1869. TAYLOR, CHARLES BELL, M.D., Nottingham.
- 1869. WEBSTER, FREDERICK RICHARD, St. Albans, Herts.
- 1868. WIBLIN, JOHN, F.R.C.S., Southampton.

. As it is very desirable that the List of Members should be kept as accurately as possible, Fellows are requested to send notice of any corrections that may be necessary to the Secretaries or to the Registrar.

GENERAL MEETING.

March 7th, 1892.

R. DOUGLAS POWELL, M.D., F.R.C.P., President, in the Chair.

Annual Report of the Council presented at the General Meeting of the Society, March 7th, 1892.

ACCORDING to custom and in discharge of its duty, the Council begs to submit to the Fellows the Annual Report upon the affairs of the Society for the past Session. There is no matter of unusual moment to announce, but the Council offers its congratulations on the health and vigour of the Society and on the progress made in all directions.

Notwithstanding the inclemency of the weather during part of the Session, the excessive amount of illness prevalent, the calls on the time of the Fellows, and the multiplicity of Societies; the meetings have been well attended, whilst the papers read and the cases brought forward have been of much interest.

The Annual Oration was delivered to a crowded meeting by Sir Joseph Lister, and the Lettsomian Lectures by Professor William Rose attracted much interest throughout the whole profession.

The Council has also pleasure in reporting that the two social functions—so long a feature of this Society—were carried out with marked success. The Annual Dinner, held at the Hôtel Métropole, was more numerously attended than on any former occasion; no less than 143 Fellows with their friends being present.

The number of Fellows now on the roll of the Society is 748; comprising 56 Life, 42 Non-Subscribing, 570 Subscribing, 33 Honorary, and 47 Corresponding Fellows. Twenty-nine new Fellows have been elected during the Session and one has rejoined. There have been nine resignations, and the Council regrets the loss sustained by the death of twelve Fellows, viz.:—Sir James Risdon Bennett, a Past President; Sir George Paget, of Cambridge; Arthur Bradford, of Bath; Dr. T. B. Christie, C.I.E., of Ealing; Dr. Charles Cogswell, for eighteen years a Trustee, and who had always been a warm friend of the Society; Surgeon-Major Edwin Drew, of Holland Park; Dr. A. T. Hickson, Notting Hill; Professor Berkeley Hill; T. Harvey Hill, Oxford, a past Treasurer of the Society; Sir Morell Mackenzie; A. S. Mackrell; and Dr. Josiah Powell.

The special features and advantages of this Society are well known to

the Fellows, and the Hon. Librarian in his Annual Reports has called attention to the increasing usefulness of the Library owing to the recent acquisition of a large number of modern books. The Council would therefore take the opportunity of urging on the Fellows the desirability of promoting the welfare of the Society, and still further increasing its usefulness and prosperity, by the introduction of new Fellows recruited from all branches of the profession.

The Council refers with considerable gratification to the issue of Vol. XIV of the Transactions. Like the preceding volume, it includes the Oration and the Lettsomian Lectures, very full reports of the proceedings, and many illustrations; but by condensation of some preliminary matter, by an improved system of recording the discussions, and by careful editing, the expenses of publication have been very materially reduced, whilst in size and matter the volume is extremely satisfactory.

The new scheme sanctioned by the Charity Commissioners for the future regulation of the Fothergillian Trust was published in Vol. XIV and has since been completed. The first award under this new Scheme will be made in 1893. These changes necessitate certain alterations in the Laws of the Society, and a Committee has, therefore, been appointed to consider the matter, and to report thereon to the Council.

The financial condition of the Society is satisfactory, but, owing to the charges incurred for the installation of the electric light and the payment of the gratuity to the widow of the late Mr. Poole, it has been decided not to draw any debentures for payment.

In consequence of considerable building operations in the immediate neighbourhood of the Society's premises, the House and Finance Committee has had several important business matters in hand, necessitating the devotion of much time and care on the part of the Chairman. Owing to private matters of pressing importance, Dr. West, fearing that he might not be able to devote himself to the affairs of the Society in the ensuing Session so completely as might be required, felt compelled to resign his post as Chairman of the Committee.

The Council has to thank Dr. West for his able and unsparing exertions in the interests of the Society during the past Session, and it has the satisfaction of announcing that Mr. Goodsall, to whom the Society is already so much indebted, has consented to again take upon himself, for a time, the Chairmanship of this important Committee.

In the last report of the Council reference was made to the impending resignation of the Registrar, Mr. Poole, and to the intention to award him a grant and pension on retirement after his twenty-five years of faithful service. Mr. Poole died on the 22nd May, 1891, and the Council, in a letter of condolence to his widow and family, took occasion to record, on behalf of the Fellows, its sense of his unfailing integrity, zeal, and courtesy, and of the kindly regard with which his memory was held by the Society.

Mr. Hall, who had occupied the post of Assistant-Registrar, has been appointed Registrar, and by his knowledge of the affairs of the Society and his unremitting attention to its interests has already proved himself a valuable officer.

In conclusion, the Council has once more to express its acknowledgments to the Treasurer, Mr. Arthur Durham, for his careful supervision of the Society's finances, and to the Honorary Librarian, Dr. Allchin, for his continued efforts in reorganising the Library, and making it more useful to the Fellows.

Report of the Honorary Librarian.

The report which I have the honour to present is again a record of the increasing prosperity and usefulness of this important department of the Society's work.

The rearrangement of the Fothergillian Trust placed at the disposal of the Library Committee a sum of about £100 for the purchase of books, of which £34 has been expended in adding to the shelves 77 volumes of standard medical works in various departments. It has been thought advisable to dispose of this sum gradually, since there is no future assured income for the Library, a circumstance much to be regretted, and which it is to be hoped will be amended by the increasing prosperity of the Society. In addition, 48 volumes have been presented, and 3 volumes received from the New Sydenham Society, making a total of 128 volumes for the year.

It cannot be said that the Library, which in most departments is fairly complete, and is, as far as possible, kept up to date, is made such use of by the Fellows of the Society as might reasonably have been expected and hoped for. The advantages of the Library, and even that books may be borrowed from it, do not appear to be as generally known as they might be; and the recommendations by Fellows for the purchase of books are extremely few.

Some progress has been made in the much-needed repair of the old and valuable works arranged in the Meeting Room, and I have much pleasure in announcing a most handsome donation from the President, which has enabled me to put the English medical works of the 16th and 17th centuries in a thoroughly satisfactory condition. A "card catalogue" of these early works is now being commenced.

The Library Committee has met three times to select books for purchase, and attend to matters connected with the Library.

It were but bare justice that I, as Hon. Librarian, should express the regret which the Society at large has felt at the death of the late Librarian, Mr. Poole. Since I have had the honour of holding the office—a period of ten years—I have annually acknowledged the very great assistance I have always received, and the indebtedness which the Society has been under to Mr. Poole, and it gives me equal pleasure in commending to the Society in the same honourable terms our present Librarian, Mr. W. R. Hall.

W. H. ALLCHIN,
Hon. Librarian.

RECEIPTS.		EXPENDITURE.	
1891, Feb. 1	£ s. d.	By Rent, Gas and Electric Light, Coals, Rates, Taxes, and Insurance	£ s. d.
To Balance from last Account	64 9 9	Repairs	478 6 0
" Life Composition	15 15 0	" Installation of Electric Light	27 2 5
" Subscriptions (including Arrears)	479 6 0	" Library Expenses	80 5 6
" Entrance Fees	33 12 0	" Salaries, Gratuities, and Collector's pound- age	17 15 0
" Rents	713 9 9	" Gratuity to the Widow of the late Regis- trar	185 11 3
" Contributions for use of Rooms	49 17 6	" Printing and Stationery	100 0 0
		" Printing Transactions, Vol. XIV	13 19 10
		" Postage, including issue of Vol. XIV	132 2 7
		" Interest on Debenture Bonds	15 1 11
		" Wages	105 6 0
		" Miscellaneous Expenses, including Refresh- ments at Meetings, Chandlery, and Cleaning, &c.	47 4 8
		" Conversazione	37 6 1
		" Balance at Bankers	51 10 6
			64 18 3
	£1356 10 0		£1356 10 0

FOTHERGILLIAN FUND.

RECEIPTS.		EXPENDITURE.	
1891, Feb. 1	£ s. d.	By Books purchased for Librai	£ s. d.
To Balance in Bank on transfer to Official Trustees, June 12, 1891	163 4 6	" Power of Attorney	33 14 6
" $\frac{3}{4}$ year's Div. on £916 10s. 5d. $2\frac{3}{4}\%$	18 18 0	" Expenses <i>re</i> New Scheme	0 11 6
		" Balance at Bankers	2 13 6
	£182 2 6		145 3 0
			£182 2 6

(Signed) ARTHUR E. DURHAM, *Treasurer.*

February 12th, 1892. Audited, compared with the vouchers, and found correct,

(Signed) T. OUTTERSON WOOD, } *Auditors.*
C. B. LOCKWOOD,

TRANSACTIONS
OF THE
MEDICAL SOCIETY OF LONDON.
119TH SESSION.

October 19th, 1891.

OPENING ADDRESS.

By the President, R. DOUGLAS POWELL, M.D., F.R.C.P.

THE PRESIDENT observed that after the numerous addresses of an introductory kind that had been given at the recent congresses at Bournemouth and in London, he would not do more than deal chiefly with matters concerning the work of the Society, making only a few digressions suggested by the work accomplished last session and promised for this. The work of the Medical Society was of peculiarly broad and catholic scope, and whilst no discovery was too new or theory too bold for its careful consideration, there was a touch of conservatism in its acceptance of new and, as yet, unestablished doctrines, due, no doubt, to the larger degree in which the practice of general medicine and surgery was represented among its Fellows. He hoped that this strong feature of the Society would be preserved, and that its Fellows would still be proportionately recruited from amongst those in general practice. After alluding to the loss the Society had sustained by the deaths of Dr. Edward Sheppard, and of their late registrar, and a brief reference to the change in the award of the Fothergillian Medal, the President referred to the fact that, in addition to their practically sufficient library of modern books of reference, the Society possessed a large collection of very valuable ancient books on medicine, which had been hitherto hidden away and unclassified, but which were well worthy of due restoration. Dr. Allchin had

already devoted much of his precious time to the work, and, with the assistance of Mr. Hall, the books were being carefully examined and prepared for placing on the shelves as rapidly as the means at disposal would permit. The President then said that it gave him great pleasure to present to the Fellows the new volume of Transactions, which had been prepared to place before the Society at the first meeting, and their best thanks were due to the secretaries for their energy and alertness. After reference to some interesting papers that had been promised for the coming session, he alluded to some that had been read during last year. The paper by Sir William Roberts on the "Deposition of Crystalline Urates in the Tissues" was a very suggestive one, and he thought that the term "uratosis," proposed by the author to describe that excess of uric acid which has been currently dignified by the more vaguely theoretical term "diathesis," was a wise suggestion. The physician was still much at a loss when asked by the student, "In what does the uric acid diathesis consist?" He thought he might speak of it perhaps as being currently regarded as a faulty chemistry of assimilation, whereby, owing to defective assimilative power, or to unduly abundant supply of material to be dealt with, products of restricted oxidation, of which uric acid was the signal type, were formed in excess. Gout was not limited to the well-conditioned any more than to the well-born, as the unhappy consequences of some fasting experiments recently conducted in public demonstrated. It would seem as though there were, if the expression might be used, a dissimilative as well as an assimilative gout. Clinically the question had an important bearing, and he (the President) hoped that able physicians in the Society who had laboured at chemical pathology in its clinical bearings would throw some more light on the matter, which was one of great importance to the welfare of the hard brain workers amongst our English people. The President then reviewed some of the advances that had been made in the treatment of tuberculosis. He expressed himself as feeling by no means so despondent as to the future of the inoculative treatment for tubercle as many of his friends. The prophylaxis of tubercle had been greatly encouraged of late years by the discovery of the organism proper to the disease. Its communicability from animals to man through the medium of milk, and perhaps of uncooked meat, had been recognised, and measures of prevention had already followed. The

President then referred to an article by Professor Tyndall in the 'Fortnightly,' based upon the recent but well-known researches of Professor Cornet on the communicability of tuberculosis. From these experiments sufficiently strong evidence had been acquired to show that the tubercle organism was not conveyed by the breath, nor indeed in any other way than one—viz., by sputum. According to this conclusion, it was certainly incumbent on every practitioner having charge of phthisical cases to review the precautions in hygiene and nursing which he might have already adopted, and to supplement them if necessary, and insist on their being carried out. They were, in fact, very simple—1. Proper spittoons only should be used, and should be carefully and efficiently cleansed, and should contain some disinfecting fluid, not powder. 2. Spitting on floors and corridors should be strongly condemned. 3. The use of handkerchiefs for reception of expectoration should be discouraged, or, if used, directions should be given for them to be thrown into a vessel containing a disinfecting fluid, and at some convenient time steeped in boiling water. 4. Floors, carpets, and furniture should not be swept or dusted, but should be wiped over with a damp cloth. These were the simple precautions that were called for in view of the direct danger they had been considering. The President added, that they had reason to thank Professor Tyndall for so trenchantly enlisting the public feeling in an important hygienic question, and that their German *confrère* deserved their gratitude for placing the facts of the matter so distinctly before them.

THE TREATMENT OF COMPOUND FRACTURES INTO JOINTS BY MEANS OF CORROSIVE SUBLIMATE BATHS.

By C. MANSELL MOULLIN, M.A., M.D. (Oxon.), F.R.C.S.

THE subject I am bringing forward this evening is not one that involves any new departure in surgery; it is merely a question of method and detail, an attempt to direct attention to a simple and efficacious plan, for carrying out general principles, upon which everyone is agreed. It is not so suitable to operations, though it may sometimes be employed with advantage in them, as to com-

4 THE TREATMENT OF COMPOUND FRACTURES INTO JOINTS

pound fractures and crushes of the limbs. Those, especially when they involve the larger joints, have always been regarded as one of the surest tests of the success of any system; if it succeeds uniformly with them its merits may be considered established, and, as a result of five years' experience of this plan, which I have used indiscriminately in cases of all kinds, I think I am justified in claiming as much for it.

There is no need to enter into any long description, the method is simplicity itself. It merely consists in placing the injured part in an antiseptic bath at the temperature of the body, and keeping it immersed. Unfortunately, from the conditions under which it must be carried out, it can only be applied to injuries of the upper extremity below the middle of the arm, and to those of the lower below the knee.

I have tried it in over thirty cases with only two failures, and these were proved to be caused by an escape of sewer gas from an old, disused, ventilation pipe. In all the rest, either the temperature never rose at all, or if, owing to delay and the occurrence of septic changes around the wound, inflammation had already commenced, it began to fall at once. Some of the cases were of the most severe description; there were two of compound fracture into the wrist joint, in one of which the scaphoid and semilunar were crushed to pieces and excised; two in which the ankle joint was opened and the bones comminuted; and three in which the elbow was so crushed that an informal primary excision had to be performed. In addition to these, there were nine of more or less extensive laceration and contusion of the hand or foot; one, for example, in which the contents of a gun, which had gone off accidentally while being carried under the arm, had blown a hole clean through the foot, and swept away a great part of the sole; another in which the hand had been so mangled in a machine that ultimately all that was left was the little finger and the stump of the thumb; and others of scarcely less extent. Only two of these were in children: all the rest were in men belonging to the ordinary class of hospital patients, the majority young, but one or two over 60 years of age.

The other group of cases, those, namely, in which the baths were not commenced until after the onset of inflammation, were scarcely less successful. In many the tissues had already begun to slough; the skin around the edges of the wound was red, and

the limb was throbbing with pain, and burning hot; but, with very few exceptions, they had hardly been in the bath for six hours before there were distinct signs of improvement. The morning temperature began to fall almost at once, and usually reached the normal by the second or third day; the evening temperature always took longer, but the descent was perfectly regular, step by step, and the inflammation in the limb above began to subside at once.

In some of these the results were very gratifying. In the last of the series, a boy, 18 years of age, who was admitted with the metatarsal bones of the foot crushed to pieces, and the skin slit down the inner side nearly as far as the internal malleolus, Hey's amputation was performed, and the foot immersed for three days with such success, that although it looked at first sight as if the part could not possibly live, the actual sloughing was limited to a very small area on the dorsum, and the temperature never rose above 100° F. In another, in which the wrist joint had been laid open and suppuration was already spreading up the tendon sheaths of the forearm and down into the palm of the hand, the progress of the inflammation was stopped at once, and the temperature fell to normal within forty-eight hours. In one only was there any serious after consequence, and this, I have every reason to believe, commenced before the baths were tried. The case was that of a man 35 years of age, whose ankle joint was run over by a tram-car: both malleoli were fractured, and the skin on the inner and outer sides of the foot lacerated and widely undermined. The wounds were well syringed out, drained, and dressed with iodoform and absorbent wool; but, in spite of this, inflammation set in, and the temperature began to rise. Baths were commenced on the fifth day, but although there was at first a certain degree of improvement, it did not continue, and, as the wound was very complicated in shape and did not drain well, it was decided to remove the astragalus. Even then the result was not satisfactory, and at length amputation was performed. The immediate cause of all the mischief proved to be a large iliac abscess, and the wound did not begin to heal, or the temperature fall to normal until this was opened. Whether it was the result of injury received at the time of the accident, or whether it was pyæmic in origin, starting from the wound before the foot was immersed, is uncertain, but at least there was no rigor, or any other evidence

or symptom of pyæmia, and after the abscess was opened there was no further trouble of any kind, the temperature never rising above 99° F.

The antiseptic I always employ is corrosive sublimate with the addition of a few drops of hydrochloric acid. Boracic acid, thymol, eucalyptus, and others that I have tried have not proved sufficiently trustworthy. The strength depends upon the size of the wound (including in this all the recesses that open out from it) and the length of time that has elapsed since the receipt of the injury. The size of the part itself, provided the skin is uninjured, does not appear material; there is no proof that any considerable quantity of the salt is absorbed through the unbroken epidermis. If the wound is foul, or if some time has passed since the injury was inflicted, I prefer to leave the part immersed for the first two hours in a solution of 1 part in 1,000, and then to change it for a solution of 1 in 10,000. If, on the other hand, the injury is quite recent, the more dilute solution may be used at once.

It is the same with the length of time during which the immersion is kept up. For fear of poisoning, at first I merely allowed the injured part to remain for an hour a day. If carbolic acid is used, even this is sufficient to render the urine dark in three or four days; but, as my experience widened and I employed this method more and more freely without meeting with any ill result, I gradually extended the time until, on two occasions, the immersion was kept up for a fortnight, without intermission, night and day. As a rule such persistence is not necessary or advisable; both these patients did suffer from mercurial poisoning; in one the gums became spongy, in the other there was diarrhœa with tenesmus; and, although the symptoms stopped of themselves as soon as the baths were changed, the immersion was undoubtedly too prolonged. In a recent case, one in which the injury is only an hour or two old, two hours night and morning are quite sufficient; but if inflammation has commenced already, and the temperature is in the least above normal, I prefer the bath to be continuous for at least the first forty-eight hours, beginning with the stronger solution; after that it may be intermittent, according to the effect produced.

The prevention of decomposition is one of the chief merits of this method, but it is by no means the only one. Pain is practically abolished; there is nothing to cause it. The temperature is

kept perfectly uniform ; no external irritant can affect the part ; there is no tension or pressure anywhere ; the limb lies motionless, equally supported on all sides ; there is no muscular spasm, for all the tissues around the wound are yielding and relaxed under the influence of the heat and moisture, and there is no inflammation. If the upper extremity is concerned it floats without any effort. No matter how extensive the laceration may be, or in what direction it lies, there is no traction on any part, for the injured tissues are so nearly of the same specific gravity as the fluid in which they lie that they are buoyed up in their natural position in the gentlest possible manner. The lower limb it is true, owing to the fact that the part submerged is relatively small, cannot float in the same way ; but if the hip and knee are flexed it rests with the lightest weight upon the sole. Even a splint is not required, although a simple wooden one, shaped to the limb, is always advisable, as it enables the part to be lifted easily out of the bath, and prevents any movement when the support of the surrounding fluid is lost.

Another most important consideration is that there is no need for the elaborate and (unless the patient is under an anæsthetic) exquisitely painful process of cleansing wounds. There is no need to pick out carefully and systematically all the dirt that has been ground in, or to cut away parts that look hopelessly injured, or to wash out the wound again and again, until, perhaps, after an hour's work, the surface seems as if it were fairly free. With baths all this is avoided ; all that is necessary is to immerse the whole injured part bodily, just as it is, after the bleeding has been stopped, and leave the dirt and the dead tissues to separate out quietly of themselves. The gunshot wound of the foot I have already mentioned is an exceedingly good example. The cavity, when the man was admitted, was filled with shot, fragments of boot and stocking, broken splinters of bone, portions of wad, and torn shreds of skin and muscle hopelessly mixed together. No attempt was made to separate them ; the foot was simply placed in a bath ; the foreign matter came away of itself ; the temperature never rose even half a degree ; and the patient's appetite never fell off or flagged in the least. With machine accidents, especially such as are met with in printers' boys, whose hands it is hopeless trying to get clean, the advantage of this is easily estimated.

In the case of burns, again, this is of very great consequence.

Nothing is more painful than the attempt to detach half-burnt clothes or to remove dressings sticking to the injured surface. Often, in doing so, unnecessary but unavoidable damage is inflicted upon the parts beneath. With children the mere sight of the preparations for dressing the wound, or even the knowledge that the time for it is come, is sufficient to make the temperature rise one or two degrees. But if baths are used the whole of this is avoided, and an enormous amount of suffering spared. I may mention that in the case of burns, owing to the large amount of surface involved, and the risk of absorption, I have only used boracic acid.

A further great merit is that the injured tissues are placed in the best possible position for recovery. In compound fractures and crushes of the limbs, where everything is torn and bruised, it is almost impossible to be certain what will live and what will not. Sometimes too much is attempted, and sloughing follows; sometimes perhaps, on the other hand, parts that might live and recover are sacrificed under the conviction that they are hopelessly disorganised. In this method it does not matter. If they die they can do no harm; as dead tissues they absorb sufficient of the antiseptic to prevent decomposition for some considerable time, and they simply remain inert and harmless until they are thrown off by the agency of the living structures beneath. If they are not quite killed they are placed in the best possible position for recovering as early as they can. There is no inflammation around; they are protected from all sources of irritation; the temperature is kept perfectly uniform; there is no tension upon any part; all the blood vessels are relaxed, and there is everything to favour the circulation through them.

There are, it is true, one or two disadvantages to contrast with this, but not, I think, of any great importance. The tissues become sodden, and the margins of the wound swollen and gelatinous; if continued too long the granulations become exuberant, and healing is delayed. The discharge collects upon the surface and coagulates, covering it with a whitish layer that makes it look as if it were sloughing; and certainly it tends to prevent union by the first intention. These results, however, are not apparent unless the bath is continued for more than three days consecutively; they are not met with if the immersion after the third day is only practised night and morning for an hour or two;

and it is not intended to apply this method to wounds which have a reasonable prospect of healing up at once.

The skin, if the immersion is very prolonged, becomes red, œdematous, and tender; and after the limb is taken out of the bath it is some little time before the blood vessels regain their tone and the tendency to œdema and passive congestion subsides. This, however, is easily overcome by keeping the part well raised when it is not in the bath, and by employing massage when the wound is sound. I have never known any excoriation produced, though, of course, a certain amount of care is required when the limb rests upon the edge of the bath. Afterwards sodden masses of epidermis may separate off, especially from the palm of the hand and the sole of the foot, but the subjacent layers soon grow hard again.

The coagulation of the albuminous secretion upon the surface of the wound is, I believe, one reason why so little of the salt is absorbed, and why symptoms of mercurial poisoning so seldom occur; but it has this disadvantage that if the wound has a very narrow outlet or is full of devious passages it tends to block them up and prevent free drainage. For this reason widely open lacerated wounds often succeed the best, and if there is only a small sinus leading into a large and complicated cavity, it is usually better to open it up freely from the first. I may mention that all incisions made after the limb has been immersed for some time bleed freely.

After the baths are left off any kind of dressing may be employed. If the wound is an open one, and the surface covered with a layer of coagulated albumen soaked with perchloride, it may be dusted over with iodoform, and kept dry, absorbent dressings, such as Tillman's wool being used, and a fair amount of pressure employed to keep in check the tendency to œdema. If, however, from the character of the injury the baths cannot be left off altogether, but must be continued once or twice a day, I have usually employed either thick layers of wet boracic lint or lint kept moist with lead and spirit lotion, and covered up with gutta-percha tissue. Either succeeds very well, and as they float off of themselves when the limb is placed in the bath, there is no trouble in removing them.

Mr. GANT had tried the plan of treating these cases by baths for a good many years, and the result of his experience was that he would not recom-

mend it as a general plan of treatment. So far as he understood Mr. Moullin, he had advised it in two sets of cases: in lacerated wounds and in lacerated wounds accompanied by fracture. Applied to the latter class of cases the treatment could not be good, owing to the liability to displacement of the fragments. Mr. Moullin had very fairly noticed the objection that the soft parts would become sodden, and the granulations œdematous. He emphasised the fact that patients submitted to treatment by continued immersion of a limb often experienced great pain; so much so that they would frequently beg that the treatment should be discontinued. He would prefer to limit the immersion treatment to lacerated wounds only.

DYSENTERY: AN ATTEMPT AT A RATIONAL EXPLANATION OF ITS NATURE AND TREATMENT.

By Professor K. N. BAHADHURJI, M.D., of Bombay.

THE pathological anatomy of the alimentary canal as studied by *post-mortem* examinations may be first noticed. The stomach and small intestines in acute and subacute cases, as a rule, show very little change beyond a somewhat slimy condition of the mucous lining; but now and again there are signs of congestion about them. In chronic cases, one often sees signs of atrophy in the fat and thin coats of the stomach and small intestines.

The large intestine presents one or more of these conditions:—

1. Red, angry-looking patches, a mass of congestion, especially in the cæcum and at the bend of the colon.
2. Simple abrasions, transverse in direction and at the lower end of the bowel there are reddish glistening groove-like patches, as if the epithelium were scraped off from the mucous surface of the bowel.
3. A simple general catarrh with thickened glands.
4. Mucous and submucous infiltration of a necrotic character, with a general thickening of the bowels, the cæcum and the rectum being very indurated and the former being often deeply pigmented. The necrotic infiltrations form what look like small abscesses or run into ulcers of various shapes and depths.
5. Areas of diffuse submucous infiltration, soft in feel and leading to gangrene of the mucous lining which sheds the sloughs often seen as shreddy masses in the stools.
6. Fulness of the portal vessels, especially of the inferior mesentery. In some very chronic cases there is a general thinning of the colon, showing marked atrophy.

The ulcers have reddish or purple margins. One sees the pro-

cess of ulceration in its different stages, there is first a congested area, circular, elliptical or transverse, the centre loses its red colour and shows a yellowish granular appearance: this spreads towards the margin. The yellowish mass, which consists of infiltration material and can be easily rubbed off with the finger, sheds its centre as it spreads, and exposes an ulcer having a greyish floor in a thickened base. The purple margins with this yellow internal granular ring are thick and overhanging; the depth varies; it may reach the muscular coat. There is generally much fibrotic thickening at and around the seat of the ulcer. Perforation now and then occurs, specially through a small round deep ulcer.

In a study of these pathological changes in the large bowels we cannot help noticing the likeness they present, to some extent, to the catarrh, the abrasions, the infiltrations, and ulcerations of the mucous lining of the mouth and the pharynx. Local or general catarrhal inflammation of the mouth is associated with glandular enlargement. Infiltration and ulceration processes about the mouth and pharynx are associated with a general swelling of the cheeks, gums, and tongue, which often shows reddish glistening patches of abrasions. In chronic pharyngitis, atrophic changes are to be seen in the uvula, the palatine arches, and the roof of the pharynx, presenting a pale, thin, wasted appearance.

A study of the signs and symptoms associated with these conditions of the mouth and pharynx will help us to fully understand the significance of the signs and symptoms in dysentery. The initial irritation gives rise to a flow of saliva. There is salivation, there is a feeling of soreness generally about the mouth and throat. The mouth and its contents feel and look rather big and full, later the salivary flow gets less watery and there is a nasty taste in the mouth and the breath smells foul. Movements of the tongue and mouth are painful, warm drinks and spirits are not agreeable, Dirty and sticky phlegm adheres to the back of the throat, and cannot be dislodged without effort. This increases as the saliva gets less and less watery. The sputa are scanty. The phlegm may show streaks of blood.

In dysentery the initial irritation in the large intestine leads to an increased watery flow of the secretions of the alimentary canal, and reflexly of its appendages, especially the liver. There is diarrhoea, often of a biliary nature, then the stools get less watery,

contain slime, and are foetid. The slime comes away with difficulty in small quantities, and at frequent intervals. One has to strain often to eject the mucus; there is tenesmus, which is only an effort of the rectum to dislodge the offending dirty mucus which is foreign to it. Slime is later mixed with blood. There is more blood from the ulcers of the large bowel than from those of the mouth and pharynx, owing to the abundance and arrangement of the blood vessels in the bowel. There is a general soreness of the abdomen, and more particularly at the cæcum and the flexures of the colon. This soreness is intensified when the irritant inflammation spreads to the peritoneal lining. Movements of the bowel cause griping pains. The stools are shreddy where ulcers are discharging sloughs, and are offensively foetid if there be any gangrenous destruction of the mucous membrane. Besides these signs and symptoms and reflex bladder trouble, such as painful frequent micturition or more often retention of urine, there are general symptoms loss of appetite, nausea, thirst and great prostration; the face wears an anxious look, the tongue is generally coated and irritable. There is slight pyrexia, with much unrest from tympanites. The pulse is soft and regular, and, as a rule, not very frequent. The skin fairly moist. In severe cases the tongue gets dry and brown, the skin dry and harsh, and the pulse loses in force and volume, and gains in frequency, and cold and clammy sweats foretell a fatal end. There are symptoms, however, which are by no means peculiar to dysentery, but common to all neglected and untreated cases of inflammatory affections and blood poisoning.

The conditions one has to deal with are thus not difficult to comprehend. To allow the catarrh to subside and the abrasions and ulcers to heal when the mucous lining of the mouth and throat is affected, one avoids all irritation. Warm drinks, spirits, and hot things are not tolerated; meat diet is not agreeable nor light enough. Moreover, meat fibres which may happen to remain in the mouth rapidly decompose and make the condition worse. Bland diet of arrowroot and milk is found very grateful and sufficiently nourishing. Alkalies or their vegetable salts taken internally improve the blood and loosen the unhealthy salivary secretion, and replace it by an healthy one. Chlorate of potash taken internally improves the secretion of the glands. Given as a mouth wash, it clears the mouth of its dirty mucus. Astringents form

useful additions to mouth washes when there is much relaxation of the mucous membrane. The stomach, which sympathises so generally with other organs in distress, the more readily does so when any functionally important portion of the alimentary canal itself, with which it has to work in unison, is out of gear. The stomach suspends its operations, so to say, to be in keeping with the requirements of the other portions of the alimentary canal, and it manifests its sympathy by its altered action, by the symptoms of the want of the usual appetite and disinclination for the usual food, both as regards quantity and quality.

Similarly in treating dysentery the points to be attended to are these: (1) All irritants, direct and indirect, should be avoided. Stimulating foods and drinks act as irritants, and for obvious reasons. All articles of food which decompose readily act as irritants. Meat and its extracts are not only useless but positively harmful. The stomach sympathises with the colon and suspends its operation to a certain extent to take off the usual strain on the colon; and the condition of the lining membrane of the stomach is much the same as the mouth and tongue, viz., it is dirty and covered with somewhat slimy mucus. Meat and its extracts are then, therefore, partially digested in the stomach, and in that condition they enter the intestines as foreign bodies, and act as direct irritants. By the ready decomposition they undergo, the unhealthy mucous lining with its dirty slime favours it greatly, they readily set up offensive and irritant putrefaction. The gases of putrefaction set up tympanites, and when absorbed along with the other products of putrefaction exert their baneful influence on the blood. The indiscriminate use of meat extracts and meat juices in cases of fever and inflammatory affections, is irrational in principle and in practice, and harmful. In all these cases appetite is meagre and digestion poor. Both by sympathy and in the natural order of things, not only the digestive but also the absorptive, the assimilative and the formative or building power, if I may so call them, of the system are fellow-sufferers, and register low. Suffering functions want rest, which means reduction of the usual work and not its increase. No doubt tissue change is great in febrile cases, as seen in the general wasting, and it is the more apparent inasmuch as the repairing and building process practically remain at a standstill till all inflammatory processes and all irritation have ceased. Our efforts

should be towards minimising this destructive tissue change as far as possible by putting the system in the best possible condition of rest and quiet. Inflammation is not over till all irritation has ceased. Till all inflammation has subsided no repair is begun. And one sees the rapidity and vigour of the building process in convalescence, *i.e.*, when all irritation and inflammation have ceased. It is absurd to try to *replace* tissue destruction by concentrated extracts or juices. One may do so, indeed if one could introduce into the circulation ready-made blood, albumen and protoplasm-juice, peculiar to each group of cells in the animal system. Given the so-called easily-digested concentrated food, what means are there to influence the absorptive power? What means are there to influence assimilation? What means are there to influence the building process? The meat juices and extracts are not nutritive but merely stimulant extractives. But suppose them, for a moment, to be nutritives, and study the results of the usual way in which they are given. 11lb. of the extracts we are told equals 20 to 32lbs. of meat. Often enough a couple of ounces or more is given in 24 hours. This means that more than 5lbs. of meat is given to the low registering power of the sick person to digest, absorb, assimilate and form flesh from. Is this not irrationally, cruelly, and mischievously overtaxing the suffering organism? Is it not like whipping a sick horse to run his fastest? When artificially digested food is given the results are no better. It is like expecting damaged machinery in a mill to turn out abundant and good production by supplying extra fine material. The large quantities thus uselessly and harmfully administered, not only overtax the system but remain as waste, which, besides acting as mechanical irritants, keep up putrefactive decomposition, the products of which act as poisonous intoxicants; they are therefore equally if not more harmful than extractives. All know how dearly one pays for gorging one's system with overabundance of meat, how the liver revolts and breaks down. And yet the sick man's sick liver in febrile and inflammatory affections is cruelly overworked with the meat extracts, and his blood is saturated with the extractives and by-products of digestion to an extent a mere fraction of which will not be borne by a healthy liver. The meat juices and extracts act as depressing intoxicants, like alcohol. What one often sees in practice is that the patient wastes away in spite of the supposed finest and

richest food so unstintingly forced down his throat. The bones project through the baggy skin, exhaustion and coma from the intoxication of the wholesale stuffing and poisoning supervene, and the patient is quietly despatched with the satisfaction of having spared nothing to nurse him and feed him every few minutes with the best obtainable concentrated juices and meat extracts. On his sick-bed he was made to eat in a week what in health he would not have eaten in a month!

Even milk by itself is irritating. It clots readily; the clots act as offending foreign bodies as much to the bowel of a dysentery patient as to that of a sickly infant. Arrowroot flour mixed with milk is the best form of diet. It acts almost as a protective to the mucous lining. It is bland and grateful, and does not readily encourage putrefactive decomposition. Stimulants in the form of drinks or even medicine will not be tolerated. Brandy, ether, ammonia, and the like will irritate the raw mucous surface of the lower bowel as much as it will burn the raw mucous lining of the mouth and throat, and make it smart. Patients taking ammonia, sal volatile, or the carbonate often complain of a burning sensation in the rectum. Alcohol irritates healthy mucous linings, and it will not spare an unhealthy and suffering mucous membrane. But alcohol we know aggravates hæmorrhoids, and it stands to reason that it will not favour dysentery ulcers which are promoted by and associated with a fulness of the portal circulation. Stimulants as administered to patients in dilutions after the half-a-whisky and half-a-nothing style are nothing more than pernicious nips which scald the patient's throat. But the complaints and remonstrances which the scalding calls forth always go unheeded!

2. The mucous lining should be helped to get rid of the unhealthy, dirty mucus sticking to it, so that it may not act as an irritant directly as a foreign body or indirectly as a centre of putrefactive decomposition. Alkalies given internally will not only loosen the sticky mucus, but will check its secretion by altering the unhealthy reaction of the blood, which presumably loses its normal degree of alkalinity in most febrile and inflammatory conditions; and alkalies do neutralise the irritating acidity of the blood, as most obviously they do in cases of fever, rheumatism and gout. Many of the bronchitis mixtures act well when they contain a little alkali; the giving of an alkali, therefore, I look upon as an important element of successful treatment.

3. Means should be taken to counteract putrefactive decomposition by acting upon the dirty mucous membrane, and nothing answers so well as bismuth subnitrate. It is slowly affected by the secretions of the alimentary canal, being not readily soluble. And this is an advantage, for if given in small and non-irritating quantities, it will reach the lower bowel and exert its influence over it locally. It moreover, soothes, gives tone to, and braces up the mucous lining generally and hence controls diarrhœa.

4. The intestinal glands themselves should be influenced, and their morbid activity, which gives out unhealthy mucus, should be stopped. This, no doubt, is done to a certain extent by improving the condition of the blood by an alkali; but ipecacuanha in small doses, and not in the irritating, empirical, and heroic doses it is generally given, has an alterative influence on the mucous membrane of the stomach and intestines. In many cases of dysentery which are treated by the large doses of ipecacuanha, real improvement has been noticed to set in and keep up with nightly administrations of Dover's powder, which contains ipecacuanha in small quantities.

5. The irritation from intestinal movement should be prevented; opium will not only check the irregular movements of the intestine due to irritation from putrefactive gases and unhealthy secretions, and thus allay pain and tenderness, but will also control the unhealthy secretions themselves, it being an important property of opium to decrease the secretion of the intestinal glands.

6. Should there be much weeping of the alimentary mucous membrane with copious watery stools, vegetable astringents are indicated. Catechu in powder form has been given with good effect. Preparations of pomegranate root also do good in some cases.

This simple method of treatment, which aims at acting on the blood, the intestinal glands, and, above all, keeping the intestinal canal and its contents "sweet," so as to enable the ulcers to take on a healthy character and heal, consists in giving a combination of bismuth, compound ipecac-powder and soda, with a dietary of arrowroot and milk. It is better to see that the ulcers have nothing irritating passing over them, rather than trying to save them the irritation from offensive contents of the colon by washing out the large bowel. It is often a good plan to give an initial dose of calomel and soda to clear away any source of irritation that may

be left lurking in the bowel. The purely astringent treatment does not meet all the requirements of the case. Hence its uncertain results. Copper sulphate is often irritating, and in many cases increases the hæmorrhage. Ipecacuanha treatment answers both when it is given in grain-dose pills, and certainly many cases improve as soon as Dover's powder is given at bedtime, as already observed. When large doses are given they often act beneficially in cases in which the emesis they induce prevents the mischief that would result from the stuffing with irritating and stimulant food, they give rest indirectly to the intestines, and allow the ulcers a chance to heal. Perchloride of mercury acts well in some cases by promoting asepsis in the bowel, but it fails in many.

A plan of treatment to be invariably successful must be rational and uniform, and based on a comprehensive view of the nature of the disease. Empiricism favours a changing plan of treatment. Empirical treatment of dysentery has recourse to ipecacuanha in large and heroic doses with alternating administrations of bismuth and morphia mixtures coupled with powerful astringent injections, and of sulphate of copper and opium pills. Perchloride of mercury both by the mouth and the rectum has also its turn in the circular empirical treatment of dysentery. Brandy and whiskey have always a free access to the patient in elastic quantities. The invariable results observed, both in private and in hospital practice, when the simple treatment of bismuth, soda, and ipecacuanha, with a dietary of nothing more than arrowroot and milk is patiently persevered in are these:—1. The patient is relieved of his uncomfortable feelings; there is less distension from gas, less griping, less tenderness, less tenesmus. 2. The quantity of blood is decreased and the stools are less offensive; their number may remain unreduced for a day or two. 3. The slime gets less, the blood disappears, and at the same time the stools become less frequent, and there is perhaps no tenesmus. There is no griping. The low diet of the patient by no means lowers him. 4. The blood has disappeared and there is very little slime, the stools are feculent and not foul, there is no griping and no distension from gas; the patient feels hungry. 5. The slime has disappeared and the stools are formed and not frequent (two to three in twenty-four hours).

On the fourth or fifth day the patient is allowed some extra milk, and, when it is found not to disagree, a little bread is allowed the next day; ordinary diet is withheld for at least a

week longer, for although the obtrusive symptoms of the ulcers subside, the ulcers are by no means completely healed, that is, covered over by the protective epithelium, and, often enough, blood reappears if ordinary diet is allowed too early.

If things are so simple, one might naturally ask whence the exhaustion and whence the severity of the disease in many cases? From its close and extensive connection with the nervous system, little affections, and especially acute affections of the alimentary canal, cause in a reflex manner an apparent loss of energy and strength, and require rest and absence of all irritation as the best restoratives. In chronic cases there is actual emaciation due to changes in the absorption tract which are against the absorption even of artificially digested food, and naturally, as a result thereof, the process of repair and tissue formation is far behind the process of tissue change and waste. In acute cases, however, prostration and exhaustion are due to other causes. One knows the depressing effects of "indigestion" gases on the stomach and intestines. One knows also the depressing and even fatal effects of the gases of putrefaction in the familiar examples of the breathing of sewer gas. In dysentery, the dirty mucous membrane gives rise to putrefactive decomposition. The open ulcer affords the gases of putrefaction every access to the circulating fluid, the seat at which the poisonous gases enter the circulation makes no difference in the blood-poisoning they induce. But more than this, the putrefaction products absorbed from wounds induce blood-poisoning quite as much when the wounds are situated on the limbs or in the mucous lining of the uterus after parturition, as in the inner surface of the intestines. There is, therefore, a septicæmic condition induced in dysentery which leads to prostration, exhaustion, and even death. And one would not be far wrong in saying that death from dysentery is due to bowel septicæmia. Anything that favours putrefaction in the disordered colon, aggravates the septicæmic blood-poisoning and renders it fatal. The readily decomposing articles of food, especially meat and its extracts, are thus positively harmful. Brand's essence smells foul if kept in a warm place, or if dirt be allowed access to it, *i.e.*, it gets putrid. In dysentery is not the condition of the colon, and the whole alimentary tract one that favours putrefaction most readily? Is it not like that of a foul dust-bin?

It will be at once apparent that I do not regard dysentery as due to any specific cause, much less to any specific germ. It is certainly not contagious nor infectious. The putrefaction effluvia from foul dysenteric stools may give rise to diarrhœa running into dysentery in those predisposed to it, much the same as other putrefaction effluvia will. The beginning of ulceration in dysentery is due to much the same general causes as is due to ulcers in the mouth and the pharynx. A lowering of the system by unhealthy water and food, and material influences, and a chronic overfulness of the mesenteric veins leading to a loss of tone and vitality of the mucous membrane, will predispose one to dysentery which is easily induced by such exciting causes as chills, a sudden change of climate (as leaving a cold country in the winter and arriving in the tropics in hot weather), a sudden change in the diet, or excesses in eating. When whole districts or collections of people, as the occupiers of "charols" (dwelling blocks of the poor) or barracks, are affected, and dysentery attacks individual after individual, it is not because there is a specific cause that is at work, but because all those affected are exposed to the same general lowering conditions as those of privation and exhausting labour.

Dysentery, then, is a simple disease with simple beginnings, but with grave endings. An ordinary wound is a simple matter, but may cost the limb, if not the life of an individual if putrefactive blood-poisoning is not avoided, or, when present, is neglected. The same obtains regarding the wound in the alimentary canal. The irritant blood-poisoning which takes place at the seat of the ulcers, and is helped in its lowering action by the loss of blood which escapes from the ulcers, if acute and severe, may take the life of the patient; or, if it be of a slow and mild character, although it may not actually kill the patient, it leaves him impoverished blood and disorganised bowel to contend against.

October 26th, 1891.

REMARKS ON FIBROUS STRICTURE OF THE COLON, WITH HISTORY OF TWO CASES DIAGNOSED BY LAPAROTOMY AND TREATED BY COLOTOMY.

By HARRISON CRIPPS, F.R.C.S.

STRICTURE may affect any portion of the colon; but I find after considerable research, both in published cases and museum specimens, that in at least three-fourths of the cases the obstruction is situated in the sigmoid flexure. The cause of the stricture is either malignant or fibrous; the former results from adenoid cancer, which forms a ring-like growth surrounding the bowel. Fibrous stricture appears to be the result of chronic inflammatory mischief, originating either in the coats of the bowel or in the fibrous tissue in its neighbourhood. Thus, ulcerations of the mucous membrane or the formation of abscess, either in the walls of the bowel or the pelvic fascia over which it runs, may be the starting point of the disease.

The diagnosis of a stricture in the large intestine at an early stage must necessarily be difficult; and, even as the disorder advances and the symptoms become more marked, it is not always easily made. Vague colicky pains with a tendency to constipation may be at first present. As the contraction increases the symptoms become decided. A rumbling of the intestines with recurring attacks of pain is observed, while there is often a frequent desire to go to the closet. Small diarrhoeic motions may be passed, or only a little discharge and wind comes away. The irritability of the bowel, so far as I have observed, seems to be greatest on first getting up in the morning. Sometimes at intervals a well-formed solid motion may be passed. This does not negative the fact that a stricture exists, for I have known fair motions passed where a subsequent *post-mortem* has disclosed a stricture which would not admit the little finger. Such motions must, of course, be formed by accumulation below the narrow part.

At the later stages of stricture vomiting occurs, but vomit of a

fæcal nature seems rarely to be present till the obstruction is complete. It occasionally happens that obvious symptoms of intestinal obstruction, accompanied by fæcal vomiting, spontaneously give way with a free discharge of fæces by the rectum. But the frequent occurrence of violent fæcal vomiting extending over a long period without fatal result, as in the first case recorded, must be very uncommon. Such attacks seem probably due to the upper opening into the stricture becoming temporarily blocked by a scybalous mass, which subsequently gives way to the churning action of the bowel.

The diagnosis between malignant and fibrous stricture of the colon must always be a matter of difficulty. Occasionally, however, an inflammatory origin is suggested by the manner in which the symptoms commence. This was especially so in Case 1. Here the sudden onset of the pain, relieved after a few days by an attack of diarrhœa, and a copious purulent discharge, left little doubt that an abscess had formed and burst, for the patient had previously been in good health and dated all her subsequent troubles from this time. If a fibrous stricture results from the healing of an ulceration, the differential diagnosis is surrounded with difficulty, for the symptoms of an ulcerated colon closely resemble those of malignant disease. Without attempting to go into the detail of the differential diagnosis between simple and malignant strictures I may mention in passing that I have found the following points of advantage:—

The duration of the symptoms may throw light on the case. Although malignant disease in its earlier stages progresses slowly it must be remembered that when it has advanced sufficiently to produce well-marked stricture, its subsequent course is comparatively rapid, and a fatal termination not far off. Indeed, it will be rarely found that a patient with cancer lives a couple of years after the symptoms of stricture become marked.

The character of the discharge helps towards diagnosis. In malignant disease it is generally considerable, while, if not at first, it soon becomes dark and blood-stained. In fibrous stricture, on the other hand, the discharge may be comparatively slight, consisting of simple mucus or muco-pus. It is true that in advanced cases where there is much secondary ulceration, the discharge may be of a coffee-ground colour as in cancer. When the stricture is due to cancer an observant nurse or patient may notice little flesh-

like fragments in the discharge, an examination of which under the microscope will at once demonstrate the presence of adenoid cancer. The general condition of the patient, as shown by weakness, cachexia, and loss of weight, must be taken into consideration, for these symptoms, though not always absent in simple stricture, form a marked feature in malignant disease.

CASE 1.—A lady, about 70 years of age, under the care of Dr. Pearson, to whom I am indebted for the following history. The patient was apparently in good health till 1883. In the spring of that year, whilst walking up a hill, she was suddenly seized with a severe pain in the left flank. The pain continued during the next few weeks, and was followed by some nausea and a sharp attack of diarrhoea and discharge. With rest and simple treatment she was convalescent at the end of a month. In October, 1883, the pain in the left flank and the nausea having recurred, accompanied by some tenderness and distension of the abdomen, she was seen by a surgeon who detected a lump low down, deeply seated in the left side. The diagnosis made at the time was that there was probably some malignant growth involving the lower part of the colon. During 1884 and 1885 she was troubled occasionally with constipation, followed by some diarrhoea.

In January, 1886, she first had what was described as a bilious attack, and since these attacks increased in frequency, and became the most prominent symptom in the case, it may be well to describe them in some detail. The patient, who had been previously feeling fairly well, with a good appetite, would suffer from constipation for some days. She would then have an uncomfortable feeling of abdominal distension, accompanied by loud borborygmi. The discomfort would gradually pass into severe abdominal pain. All appetite would be lost, and she would have an intense feeling of nausea, and this would be followed in a short time by severe vomiting, lasting on and off for twenty-four hours. The vomited matter would consist almost entirely of fluid faecal material. Generally at this time she would have two or three copious diarrhoeic stools, the matter passed by the rectum being similar to that passed by the mouth. The attack would then pass off, the appetite return, and she would remain in fairly good health until another occurred. At first there was an interval of two or three months between these attacks, but their frequency gradually increased, so that latterly the intervals were not more than a fortnight or three weeks. The spasmodic pains also became more intense, and the patient's general condition was very grave.

On being asked to see the patient by Dr. Pearson in July, 1888, I found her in a very feeble state after one of these attacks. She was naturally a stout woman, and though she had become much thinner, there was so much fat in the abdominal walls as to make an examination of the abdomen difficult, but, on deep firm pressure, there appeared to be some indistinct fulness in the left groin. An examination by the rectum and vagina gave a negative result. The general symptoms and character of the vomiting suggested so strongly a stricture of the large intestine that an exploratory operation was advised and consented to.

On July 19th, distension of the abdomen having again recurred, and seeing that she was evidently on the brink of another attack, assisted by Mr. Bowlby, I opened the abdomen on the left side by an incision 3 inches

long, midway between the umbilicus and anterior superior spine. An enormously distended coil of the sigmoid flexure immediately presented itself. Upon tracing this downwards towards the brim of the pelvis, it was felt to end abruptly in a firm, hard, pipe-like piece of bowel about as thick as the thumb; this was tightly bound down to the brim of the pelvis by dense fibrous tissue. It was at once obvious, on this examination, that, owing to the dense fibrous tissue into which the coats of the bowel had been converted, any operation on the strictured part itself was impracticable. The distended coil above the stricture was therefore drawn out as far as possible, and an inguinal colotomy performed after the manner I have already described in previous papers. The piece of gut was so distended that it would not have been safe to leave it unopened, for any vomiting would almost certainly have caused its rupture. It was, therefore, after having been very accurately stitched to the parietal peritoneum and skin, opened. Several pints of pea-soup-like fæces flowed out. A stream of water was kept pouring over the wound until the bowel had completely relieved itself; the dressings were then applied. The wound healed without any trouble. In a few months the artificial anus thus formed worked with fair regularity. The patient's general health rapidly improved, and since the day of the operation she has never had the slightest return of fæcal vomiting, nor any of her old symptoms. Occasionally, when there is diarrhoea, the artificial opening gives some trouble, at other times it can be kept clean.

The patient has become very stout, and there is some tendency for the bowel to prolapse, but it can be replaced without difficulty, and kept in position with a pad. The patient is now (more than three years after the operation) able to drive and walk out daily.

CASE 2.—Mrs. G——, aged 53, brought to me by Dr. Goodchild, had enjoyed fair health till May, 1888. At that time she often used to notice that she would be suddenly seized with violent pain in the abdomen. She would want to pass a motion, but only a little wind would come away. As the summer advanced the bowels became very irregular, sometimes being constipated, at others relaxed. There was a frequent desire to relieve them, but generally only a little mucus would pass. During the autumn and winter she gradually became worse. At times the pain would increase, and she would pass a considerable amount of mucus. This was occasionally mixed with a small quantity of blood. At this time she had a relief from the bowel every day, or every other day.

On March 8, 1889, I saw the patient for the first time, and made the following note of her condition: On first getting up in the morning she had a desire to go to the closet, and passes a teaspoonful or two of discharge. This sometimes occurs at other times of the day, but not often. She cannot pass wind without fear of the discharge coming away. The motion is generally relaxed, but sometimes well formed. Often she has intense colicky pains in the bowels, in the intervals being free from pain. Upon examination the abdomen was very large, flat, and flabby, and nothing abnormal could be felt. An examination by the rectum disclosed nothing but some large loose folds of mucous membrane. I advised injections and a strict diet. The patient felt a little better at first from the treatment, and was able to come downstairs, but was often driven up again with the pain. In the morning she could not stand up without mucus passing involuntarily away, so that she had to dress

sitting. The pain would come on every two days. One day she would be quite free, and the next in great pain. Occasionally, for a week or two, she would feel much better; her appetite was very bad, and she was getting markedly thinner. The motions were small, and passed in little pieces.

On August 25th, 1889, she took a dose of castor oil. This brought on violent pain and vomiting, and the abdomen became so tender that the weight of the clothes could scarcely be borne. Owing to this pain she readily consented to an exploratory operation, with a view to colotomy if obstruction was found. On September 1st, in the presence of Dr. Goodchild, and with the assistance of Mr. Balgarnie, I opened the abdomen in a manner similar to the last case. On passing my finger into the abdominal cavity some hard, scybalous masses could be at once felt in the sigmoid flexure. The bowel could be traced down to the pelvic brim. Here, and for a distance of two inches above it, it was tightly strictured, feeling like a hard cord bound down to the fascia behind. The bowel immediately above the stricture was dilated. This was traced upwards till a healthy portion could be found, which was drawn out and sutured to the peritoneum and skin, and opened the following day. The patient convalesced without trouble.

At the present time, two years after the colotomy, she states that since the operation all symptoms have been completely relieved, and she now considers herself in excellent health, and is very satisfied with her condition. The new anus, as a rule, acts but once a day. She has no difficulty in keeping herself clean, and is able to take walking exercise without discomfort or inconvenience.

In the first of the two cases recorded the symptoms were most pronounced, and seemed to be more consistent with a stricture low down in the large intestine than with any other disorder. The second case was not so clear, though the probability of a stricture was suggested. The gravity of the symptoms in both cases was such as to make it apparent that without relief a fatal termination could not be far off. In these circumstances it appeared that the right thing to do was first to find out what was the matter, and the only way in which this could be done with certainty was to look into the abdomen to see. It was conceivable that if an obstruction were found it might be due to some cause which surgery could remedy; and, if such was not the case, by performing a colotomy above the obstruction death might be averted, and the patient's life made comparatively comfortable. The site selected for exploration in each instance was on the left side, between the umbilicus and the anterior superior spine. In the first case, the hardness that could be felt on deep pressure in this place was taken as an indication of the right spot for the incision. In Case 2, where nothing could be felt through the abdominal walls, it was selected from the fact that when there is nothing to

indicate the site of stricture the chances are strongly in favour of its being either in the sigmoid flexure or in the upper part of the rectum. Another advantage of the exploratory incision being in this neighbourhood is that, if the obstruction is found to be irremediable, or an anastomosis impracticable, an inguinal colotomy can be completed without further trouble, as in my two cases.

Had the obstruction not been thus found, I should have enlarged the incision sufficiently to have admitted the hand and explored the rest of the colon. If the stricture had proved to be in the transverse or descending colon the exploratory opening might have been closed, and colotomy performed on the opposite side.

It would be out of place in this paper to dilate on the merits and advantages of inguinal colotomy; but I must state that with an experience of nearly 100 cases I have not altered my views as to its great superiority over the lumbar method. Moreover, the two cases recorded are additional evidence of its value, for the abdominal incision afforded the means of confirming the diagnosis, and then was used for the completion of the operation.

In conclusion, I would ask physicians whether in these cases they could help us to a more precise diagnosis as to the presence of stricture, as to its nature, and the point where it is likely to be found; so that when an exploratory operation is undertaken, the surgeon may confidently expect to find a condition admitting of relief.

FIFTY CASES OF LEFT INGUINAL COLOTOMY. WITH REMARKS ON THEIR POINTS OF SPECIAL INTEREST.

By HERBERT W. ALLINGHAM, F.R.C.S.

IN giving an account of my first list of fifty cases of left inguinal colotomy, it is my intention not only to record the successes, but also to point out what I consider to have been defects, in the operation of inguinal colotomy as practised by me. I shall also show that the experience I have gained from these cases has enabled me to see how to remedy these defects.

I have further performed several right inguinal colotomies, but I do not propose to include those in this list. The same remark

applies to my cases of lumbar colotomy. I have not performed this operation in many instances, for from the result of my own cases and of the cases I have seen under the care of other surgeons, I consider lumbar colotomy to be far inferior, generally speaking, to inguinal, both from the surgeon's point of view, and as regards the present and future of the patient. I am aware that the advocates of lumbar colotomy have published many cases, and that they have never admitted that there are any drawbacks whatsoever in the operations.

To me this is unaccountable, for from my own cases and others which have come under my notice, it is plain that lumbar colotomy is attended with many disadvantages. These defects must have been noticed by other operators, but they have been ignored entirely, and the performers of lumbar colotomy have confined themselves to an unstinted laudation of their own method.

Statistics compiled and records published in such a manner can surely be of little or no value, for the only way to arrive at any true and valuable inference is to admit freely both the advantage and disadvantage of respective operations, to weigh them together, and to decide in favour of that method which has the least defects and the greatest merit; thus only can patients be treated in the best and soundest manner.

There is no doubt that lumbar colotomy, as well as inguinal colotomy, possesses its own peculiar advantages, and is of excellent application in certain conditions for which it is fitted. But the choice between these two operations does not lie within the scope of this paper; it will receive full treatment in other works on the subjects which I have now in hand.

I now turn to my own immediate business, inguinal colotomy, and various points connected with the operation, which I shall discuss in the order indicated upon the diagram of cases, and these are :

1. The nature of the disease.
2. The ordinary inguinal colotomy as I perform it, and the occasions for the use of the supplementary operation which I have previously advocated in the 'British Medical Journal.'
3. The length of the sigmoid mesentery.
4. The spur.
5. Prolapse after the operation from the upper or the lower end, or from both ends.
6. The action of the bowels, and from which end.
7. The result, that is, the duration of life after the operation.

1. It will be noticed that in my 50 cases, malignant disease was by far the most frequent cause that necessitated the operation, for to it 41 cases were due; the remaining 9 cases were operated upon for ulceration with stricture, often combined with fistula so commonly associated with those diseases.

2a. *The operation* as I perform it. The distinctive feature of my own method is the extreme importance I attach to the procuring of a good spur, so as to prevent fæces passing beyond the inguinal opening into the rectum, and thus further irritating the diseased condition for which the operation is done. This question of the spur will be treated of under my fourth head.

I will now give a brief account of my method which I have so often described in detail. An incision not more than 2 inches in length is made about $1\frac{1}{2}$ inch inside the left anterior superior spine of the ilium. This I have found from anatomical research to be the best spot. The abdomen is opened, and a sponge is then introduced to keep the intestines and omentum from prolapsing, while the parietal peritoneum is sewn to the edges of the skin wound. This being done, a good loop of the sigmoid is drawn out (Fig. 1), and a stitch is put through the skin on one side, then through the mesentery behind the bowel, back again through the mesentery, and is then tied to the end of the suture which has passed through the skin. When this is tightened, it keeps the



FIG. 1.

peritoneum of the mesentery against the parietal peritoneum and quickly adheres; thus the gut is prevented from slipping back. Then in several places around the gut is fixed by the passage of

sutures through its peritoneal and muscular coats to the skin. This is one of the simplest of all the forms of operation that have been proposed, and can be easily performed in fifteen minutes.

The gut next day is opened, or, if there be no distension, it can be left for three or four days, according to the condition of the patient. In a week's time, if the walls of the divided gut be too prominent, they can be cut off with scissors flush to the skin.

2b. The supplementary operation, as I term it. This is done to prevent prolapse of the gut through the opening, which may occur every now and then, and, if the patient is likely to live long, may be a source of great annoyance and discomfort.

It will be seen that I had performed my first eight cases before I began to appreciate that there was a great danger of prolapse, and that then I commenced to devise this supplementary operation.

Prolapse is undoubtedly due to a lengthy mesentery which allows the gut above and below the inguinal opening to prolapse from its upper or the lower end, or even from both ends. The gist of my further operation is this: When the belly has been opened and the parietal peritoneum has been stretched to the skin, the gut is drawn out and is pulled upon till it is taut above, and, similarly, till it is taut below; in other words, I pull out all the slack portion of the gut, which, in cases when the mesentery is lengthy, may amount to many inches. All the portion that has been drawn out is then fixed outside the belly, as is done in the ordinary operation (Fig. 2). In two or three days the gut is opened so as to allow the exit of wind, and, in a week or so, all the gut outside the



FIG 2.

belly is clamped with my spiked clamp close to the skin, and the portion above the clamp is cut away (Fig. 3). In some cases the

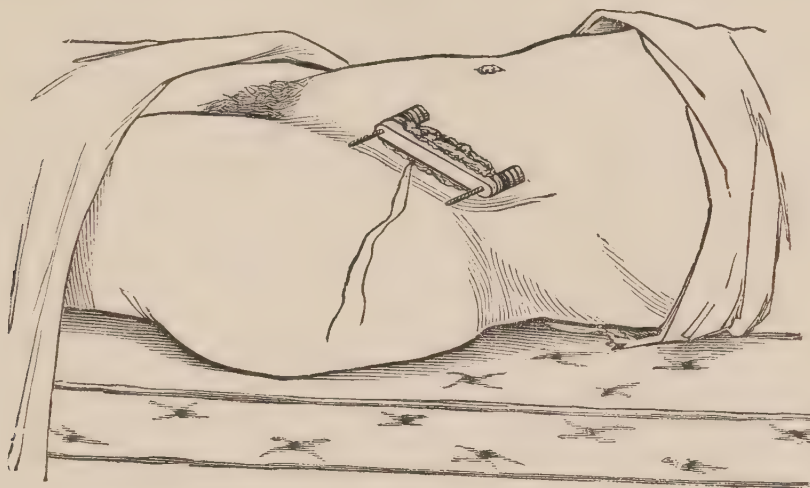


FIG. 3.

part removed has been a foot long and has weighed more than 6 ounces. The clamp is left on for twenty-four hours, and is then removed, no bleeding occurring.

It will be noticed that in Cases 9 to 13 I did this operation in all instances, whether for malignant disease or for non-malignant strictures. I then began to think that this proceeding was rather too severe in advanced cases of cancer, in which the patients had not long to live, and any prolapse was therefore of minor importance. In such cases I grew to hold it unnecessary to subject patients to any more pain and trouble than was absolutely required. Such considerations led me to reserve this supplementary procedure for cases of simple stricture where the patients might live for years, and it was important to prevent any discomfort and more or less constant prolapse.

Still, inasmuch as my clamp does away with any risk of hæmorrhage, I am not afraid to perform this supplementary operation in selected cases of malignant disease. Out of my 50 cases, 14 have been treated in this manner with perfect success as regards any after-prolapse; out of these, 7 were performed for malignant disease, 3 of them being done before I considered the question, and the other 7 for ulceration with stricture. I may note that in spite of the supposed danger of this supplementary operation not one patient died from it.

3. *The Length of the Mesentery.*—For purposes of description

and classification I divide mesenteries into long, medium, and short; by long, I mean cases in which the mesentery connecting the sigmoid with the iliac fossa is at least 5 inches in length, or even more. In such cases there may be some difficulty in finding the gut from the inguinal opening, but I myself have never experienced any trouble, for I pass my finger up towards the kidney where the gut is fixed, or down to the rectum, where it is also fixed. I thus trace it up or down to the sigmoid. In 17 of my cases the mesentery was long. By a medium mesentery I mean one the length of which is at least 2 or 3 inches, so that it is possible to pull the gut well out of the wound, and to make a good spur; there were 25 of this class of mesentery. By short I designate cases in which there is practically no mesentery at all, and it is therefore difficult to fix the gut to the skin. Of even more importance is the circumstance that there is no possibility whatsoever of passing a needle behind the gut and so forming a good spur; indeed, no spur can be made. Thus the patients are left in a miserable condition, for some of the fæces pass beyond the opening in the inguinal region towards the growth. Further, in patients who are fat it is very difficult to fix the gut to the skin, and even if the gut has been fixed, any vomiting or coughing may tear away the sutures, and the gut may slip back into the abdominal cavity. This actually occurred in Case 21. There were eight instances of a short mesentery.

4. *The Spur*.—To procure a spur means to fix up the gut (by the mesenteric stitch) in such a manner that no fæces can possibly pass from the upper part of the intestine beyond the inguinal opening into the portion of the gut below the opening. Such passage of fæces will only further irritate the malignant growth or innocent stricture. Unless such a spur has been obtained, I consider the operation to have been a failure. This is particularly the case at the present time, when inguinal colotomy is done much earlier than formerly, and when one of the main objects of the operation is to relieve or allay this very irritation. If through neglect to make a spur this irritation is maintained or even aggravated, and the concomitant diarrhoea and pains are not stopped, we shall merely have added to the patient's discomfort, for he will have a fæcal fistula in the groin instead of a complete and perfect artificial anus intended to relieve the irritation of the rectum below the opening. This matter is so exceedingly important that

I must be allowed to distinguish clearly between a fæcal fistula and an artificial anus.

A fæcal fistula is an opening into a piece of gut communicating with the surface of the body from which fæces issue, but at the same time some of the fæces pass beyond the fistula into the distal portion of the gut. An artificial anus is an opening in which all the fæces pass through the opening on the surface of the body, and none whatever pass into by the distal portion of the gut.

Now, if inguinal colotomy is performed and no spur is made, we have a condition of fæcal fistula, for fæces pass both by the inguinal opening and also into the distal portion of the gut. When, however, a spur is made, all the fæces pass through the opening in the groin, and more can enter into the distal end of the intestine; thus any fæcal irritation of the growth is entirely prevented.

I have tried to put this matter in a clear light, because some surgeons deny the necessity of making a definite spur, and therefore, in my eyes, their operations fail in an exceedingly important point. It will be observed that in 9 out of my 50 cases, no spur was formed. In my first 3 cases I had not come to appreciate the importance of the spur, and therefore did not attempt to make one; in fact, I did not use the mesentery stitch. In the other 6 cases, namely, Cases 21, 22, 28, 37, 42, and 45, the mesentery was of the short variety, and no spur was procurable. In these instances the patients' anatomical peculiarities were the reason of their suffering discomfort from fæces occasionally passing *per rectum* as well by the inguinal opening, and thus causing pain and irritation.

5. *Prolapse from the Inguinal Opening.*—This may occur either from the upper end of the gut, that is, of the part which is continuous with the descending colon or from the lower end, that is, of the part leading to and continuous with the rectum; sometimes, indeed, there may be prolapse from both ends at the same time. I have already observed that it is of far more importance to prevent this condition when patients are likely to have a fairly long lease of life, and it is on that account that I devised the supplementary operation already described. Now we know (and my own cases lend corroboration) that prolapse occurs only when there is a long mesentery which enables the gut to intussuscept through

Fifty Cases of Left Inguinal Colotomy.

No.	Name.	Nature of disease.	Supple- mentary operation.	Length of Mesentery.	Spur.	Prolapse from upper or lower end or from both.	Action of bowels from upper or lower end.
1	Mrs. T. C. . .	Stricture, ulceration, and fistulæ	No	Medium	No	No	Upper and by rectum.
2	Mrs. A. H. . .	Malignant disease	"	"	"	"	"
3	Mrs. E. C. . .	"	"	Long	"	From upper and lower end	"
4	Mr. I. J. . .	"	"	Medium	Yes	No	"Upper.
5	Mrs. B. J. . .	"	"	"	"	Slight from upper and lower	"
6	Mr. F. C. . .	"	"	"	"	No	"
7	Mrs. S. C. . .	"	"	Long	"	Slight from upper	"
8	Mrs. F. G. . .	"	"	"	"	Slight from lower end	"
9	C. S. . .	Stricture and ulceration	Yes	"	"	No	"
10	Mr. C. B. . .	Malignant disease	"	Very long	"	"	Lower.
11	Miss A. T. . .	"	"	Long	"	"	Upper.
12	K. T. . .	Stricture, ulceration, and fistulæ	"	"	"	"	"
13	Mr. A. Z. . .	Malignant disease	"	Very long	"	"	"
14	Mr. W. M. . .	Malignant disease of rectm. & bldr.	No	Medium	"	"	"
15	A. D. . .	Stricture and ulceration	Yes	Long	"	"	"
16	S. R. . .	Malignant disease	"	Medium, adherent omentum	"	"	Lower
17	D. C. . .	Stricture, fistulæ, and ulceration	"	Long	"	"	"
18	M. H. . .	Malignant disease	No	Medium	"	Slight from upper and lower	Upper
19	K. G. . .	"	"	"	"	No	Lower
20	J. T. . .	"	"	"	"	No	Upper and by rectum.
21	S. W. . .	Stricture and ulceration	"	Very short	No	"	"
22	W. F. . .	Malignant disease	"	"	Yes	"	"Upper.
23	A. H. . .	"	Yes	Medium	"	Upper	"
24	Mr. R. B. . .	"	No	"	"	No	Lower
25	Mrs. J. W. . .	"	Yes	"	"	Lower	Upper
26	Mrs. T. J. . .	"	No	Long	"	From upper and lower	"
27	Mr. T. D. . .	"	"	"	"	No	Upper and by rectum.
28	Mr. B. L. . .	Malignant, obstruction, paralysed	"	Short	No	"	Lower
29	Mr. G. A. . .	Malignant disease	"	Medium, adherent omentum	Yes	Slight upper and lower	Upper.
30	T. B. . .	"	"	Medium	"	No	"
31	M. G. . .	"	"	"	"	"	Upper and by rectum.
32	N. O. . .	Malignant disease, obstruction	"	Short	"	Lower very bad	Upper.
33	R. H. . .	Malignant disease	"	Long	"	No	Upper and by rectum.
34	T. D. . .	"	"	Very short	"	Slight from lower	Upper.
35	N. T. . .	"	"	Medium	"	Upper	"
36	E. R. . .	"	"	"	"	No	Upper and by rectum.
37	Mr. M. R. . .	"	"	Short	No	"	"

No.	Name.	Nature of disease.	Supple- mentary operation.	Length of Mesentery.	Spur.	Prolapse from upper or lower end or from both.	Action of bowels from upper or lower end.
38	Mr. J. C. ...	Malignant disease	Yes	Long	Yes	No	Upper.
39	Mr. T. P. ...	"	No	Medium	"	"	"
40	Mr. G. T. ...	"	"	"	"	"	"
41	M. D. ...	Stricture and ulceration	Yes	Long	"	"	"
42	M. G. ...	Malignant disease	No	Very short	No	"	Upper and by rectum.
43	A. D. ...	Stricture, tuberculous (?)	Yes	Medium	Yes	"	Upper.
44	Mr. K. B. ...	Malignant disease	No	"	"	Slight from upper	"
45	Mr. C. P. ...	"	"	Very short	No	No	Upper and by rectum.
46	E. P. ...	"	"	Medium	Yes	"	Upper.
47	E. C. ...	Stricture and ulceration	Yes	"	"	"	"
48	C. A. ...	Malignant disease	No	Long	"	Slight from upper and lower	"
49	Mr. T. A. ...	"	"	Medium	"	Slight from upper	"
50	Mr. T. G. ...	"	"	Long	"	From lower	"

the part of the gut which has been fixed, that is, sewn to the belly wall. I arrived at this conclusion from noticing that where there was a short mesentery there was no prolapse. Again, whenever I had performed the supplementary operation, that is, whenever I had drawn out and removed the slack portion of the gut, there was also no prolapse.

To obviate this prolapsed condition, Mr. Cripps has advised that the gut should be pulled down until it is taut upon the upper end, and that all the slack portion should be returned into the belly, and then the gut be stitched up to the skin wound. No doubt this is a good method, for there can then be no prolapse from the upper part of the gut. Nevertheless this plan does not prevent prolapse from the lower part of the intestine where the mesentery is long. However, the suggestion is of much value, and should always be carried out in malignant cases when the supplementary operation is not advisable. Lastly, I have seen prolapse occur from both ends at the same time, not only in my own cases but in those of others.

Prolapse took place in 17 out of my 50 cases; in 5 out of the 17 from the upper end alone; in 6 out of the 17 from the lower end alone; in 6 of the 17 from the upper and lower end together. In all these cases the mesentery was either long or medium in length, though the prolapse did not occur in every instance of a medium-sized mesentery. Further, prolapse did not take place in any case when the supplementary operation had been performed in spite of the great length of the mesentery in a large number of these instances.

6. *The Action of the Bowels whether from the Upper or from the Lower End of the Double-barrelled Opening resulting after my Operation.*—Though the circumstance may seem strange, still in 7 of the 50 cases the bowels acted from the lower of the two orifices. This is to be explained by the fact that the gut was twisted when it was pulled up into the inguinal opening, and fixed there.

In most of these cases the mesentery was long; in some cases it is reported to have been medium, but it may have in reality been long, but have been rendered of medium length by the twist. This twisting is curious, but it is also important from an operative point of view. For some surgeons have suggested division of the gut, to be followed by dropping the distal end into the belly, and

fixing only the proximal end to the skin. Now if the gut is twisted, the operator may sew up the upper end and drop it back, fixing up the lower end in the belief that it is the upper end. Needless to say, such a mistake would result in the patient's death. Such a case has actually come within my notice.

The importance of the spur is further instanced in this matter of the action of the bowels. In Cases 1, 2, and 3, in which I did not attempt to make a spur, and in those other cases in which the mesentery was so short that no spur was attainable, the bowels acted from the inguinal opening and *per rectum* also, namely, in Cases 21, 22, 28, 32, 34, 37, 42, 45. Thus in 11 cases the patients were occasioned some distress, and the operation, therefore, was not a complete success.

Out of 50 inguinal colotomies, 36 inguinal ordinary operations, 14 supplementary operations; 17 mesentery was long, 25 mesentery was medium length, 8 mesentery was short; 9 cases was no spur; 17 prolapse took place; 5 out of the 17 from upper end alone, 6 out of the 17 from lower end alone; 6 out of the 17 from both ends together. The gut was twisted when fixed up, so that the bowels acted from the lower of the two openings in 7 out of 50 cases.

7. *The Result of the Operation and the After-Duration of Life.*—The results of inguinal colotomy may be divided into several classes. If the patient lives for fourteen days after the operation he may be said to have recovered from the effect of the operation pure and simple, as in inguinal colotomy generally the wounds heal quickly. As a matter of fact, the duration of life is very difficult to ascertain, for patients often go away, and there is therefore no possibility of learning whether they have died speedily or have lived for a considerable period. Applying the fourteen days' limit, I have lost only 2 cases, namely, Cases 21 and 32.

In Case 21 the mesentery was very short, and there was great difficulty in fixing it to the skin. Moreover, a few days later the patient, who was very fat, developed bronchitis. In a fit of coughing some bleeding took place from the wound, and the house-surgeon, who was unable to find the bleeding vessel, applied pressure; thereupon, from the pressure and the coughing combined, the gut was torn away from the inguinal opening and fell back into the belly. Fæces then escaped into the abdominal cavity, and in a few hours the patient died of peritonitis.

I now think that in Case 32 it would have been wiser to have done lumbar colotomy. From the five weeks' obstruction the intestines were greatly distended; hence from the distension of the large intestine it was next to impossible to get the gut well to the surface from the small inguinal opening. Further, the distension made it almost impracticable to sew the gut to the skin without perforating the mucous coat, and thus allowing flatus to leak into the peritoneum. Fortunately I escaped this misadventure; but after the operation, in consequence of the patient vomiting, the small intestines in their distended state were forced through the wound, tearing away the sutures which fixed the large intestine to the inguinal incision.

In these cases of great distension it is imperative to open the gut at once, and thus there is a danger of fæces finding their way into the peritoneal cavity. Now when lumbar colotomy is performed in such cases, the gut is pushed into the loin, and the posterior part, which is uncovered by peritoneum, is freely exposed by the distended gut separating its peritoneum at the back part. Thus the intestine can be opened on its non-peritoneal surface without any dread of the peritoneum being damaged, and as soon as it has been opened and relieved of its distension it can be drawn to the loin and stitched there.

I have now pointed out all the difficulties which I have experienced in these my first 50 cases of inguinal colotomy. I have reason to hope that my observations and inferences may be of interest and of practical use to others.

The PRESIDENT said that the diagnosis of the seat of the stricture was often a very difficult matter, in many instances an exploratory incision being necessary to decide both its seat and nature. On palpation the labouring and hypertrophied bowel could often be recognised behind the seat of constriction, and was an aid to diagnosis, and in one case of stricture of pylorus which had been recently under his care in the Middlesex Hospital he had observed reversed peristaltic action very distinctly. He inquired if such a phenomenon had been often observed by the authors.

Mr. BOWREMAN JESSETT congratulated the authors on the success of the operation reported by them. In respect to Mr. Cripps's remarks, he must say that without the surgeon was quite sure of his diagnosis as to the seat of the obstruction, he preferred the median incision, as by that means the whole abdominal cavity might be explored, and if found necessary to perform an inguinal colotomy on either side it only meant another incision at the seat of operation, which was not of much consequence. With regard to the method of operating for inguinal colotomy as propounded in this room some few years ago, and still practised by him now, he (Mr. Jessett) would like to make a few remarks, as for some time

past, after the experience of a large number of cases, he had introduced some not unimportant innovations. Firstly, in the treatment of the parietal peritoneum, the method of stitching this membrane to the skin he had entirely given up, as in his opinion it was absolutely needless, indeed, he believed harmless, as it prevented the direct union of muscle to muscle and skin to skin at the ends of the wounds, which caused a weakness and possible hernia at that point. Secondly, in passing the double suture through the parieties and meso-colon to sling the loop of intestine, he found that the suture cut into the skin, causing considerable pain and forming a nasty ulcer. To overcome this he was in the habit of using two bone bars, about $1\frac{1}{2}$ inches long, with two holes drilled through, through which the sutures were passed and tied. This enabled him to do away with all sutures for forming the serum and muscular coats of the intestines to the parieties, as the pressure of these bars was quite sufficient to keep the parietal and visceral peritoneum in such close contact that there was, in his opinion, absolutely no fear of any hernial protrusion of either the small intestine or omentum. He was only in the habit of passing one extra, or, at the most, two, at each end of the wound for the union of the parietal wound beyond the space occupied by the loop of intestine. Thirdly, on removing the loop of intestine, he did not think the clamp shown by Mr. H. Allingham at all necessary; indeed it caused a good deal of pain, and in his experience he had never had any trouble in picking up the vessels as they were divided. There was no pain in dividing the intestine, and very little in the division of the meso-colon. Fourthly, in certain cases, such as the case from which the specimen he exhibited was taken, he thought it decidedly of advantage to sever the inguinal flexure close, and drop the lower end, and to fasten a tube into the upper portion, to which he had in several cases attached an india-rubber tube, which was conducted to a vessel containing solution of carbolic acid beneath the bed, very much in the same way that Mr. Paul, of Liverpool, had recently described in the 'Lancet.'

Mr. MORGAN remarked that the Society was to be congratulated on the two papers which had been read that evening, which would be of the greatest interest to surgeons, and it was to be regretted that some of those who had so strenuously defended the value of the lumbar operation were not present to hear such important testimony regarding the revived inguinal operation as had been furnished that evening. He had in the year 1888 to deal with a case almost similar to the two related by Mr. Cripps. A lady who had frequently suffered from recurrent attacks of pelvic cellulitis, and who for a considerable period had been troubled with obstinate constipation, became suddenly very seriously ill. When seen in consultation she was suffering from great distension of the abdomen, hiccough, and all the symptoms of commencing peritonitis. She had not, however, shown the symptoms that Mr. Cripps had described. Her condition was so critical that an exploratory incision was made, and there was found to exist a large amount of cicatricial tissue which had constricted the lower part of the colon and sigmoid flexure. The bowel was attached in the ordinary manner and, owing to the urgency of her condition, was opened on the following day. She experienced immediate relief, and she is now alive and well. Mr. Morgan asked whether Mr. Cripps would be inclined to extend the scope of his suggestion so as to include those cases of obstruction in which the situation of the constriction could not be guessed. In one case of such doubtful nature Mr. Morgan had performed a median laparotomy and by this means had ascertained the

existence of a malignant growth in the descending colon, for which a lumbar colotomy was then and there performed. In another case of great obscurity he had reason to regret not having resorted to this means of diagnosis. It was that of a man, aged 51, who gave evidence of partial but not complete obstruction in some part of the large intestine, apparently not the descending or transverse colon. After being watched for some time, his symptoms suddenly became acute, and colotomy was performed in the right inguinal region. The relief was immediate, but he died shortly after, and it was found at the *post-mortem* examination that there was a narrow ring of epitheliomatous growth in the ascending colon, a very little above the artificial opening, which, if detected in the manner suggested, might easily have been removed. Time did not permit Mr. Morgan to comment on all the points of Mr. Allingham's paper, but, although he had performed a large number of inguinal colotomies and had followed carefully the suggestions in Mr. Allingham's writings, he was still in doubt as to the best plan of avoiding the troublesome after consequences in the shape of prolapse, which, even by Mr. Allingham's tables, were by no means infrequent in his own cases.

Mr. GOODSALL pointed out the advantages of inguinal as against lumbar colotomy. He said that upwards of 80 per cent. of strictures in men were malignant, whereas less than 20 per cent. of those in women were. In fibrous stricture, the bowels acted much more frequently than in malignant cases. In cases of non-malignant stricture in women there might have been a history of one child or of one bad mis-carriage, but they were usually sterile. A colotomy opening placed on a level with, and one inch to the inner side of, the anterior superior spine of the left ilium made it less likely that the fæces would pass on to the rectum, in cases in which only an opening had been made in the colon.

Mr. BRUCE CLARKE remarked that whatever precautions were taken in the operation of inguinal colotomy, there would be some cases in prolapse of the gut which would prove troublesome features later on in the case, but it did seem to him that seventeen was a very large number of cases out of a total of fifty in which to get such a complication. He could not help feeling that the very means which Mr. Allingham took to procure a spur were possibly the cause of subsequent prolapse. For his part he had operated on some dozen cases, and had assisted his friend, Mr. Cripps, at numerous others, and he had never found it necessary to remove a considerable piece of sigmoid flexure at the time of operation, and though he could not off hand state how often he had observed prolapse, yet certainly not in 34 per cent. of his cases. The plan he adopted was much the same as that employed by his friend, Mr. Cripps; he sutured the back part of the intestine to the abdominal wall, after having pulled down as much of the intestine as he could get hold of. He then fixed the sigmoid flexure in such a way that the fold which eventually formed the spur, came as nearly as possible at the lower margin of his wound. By this plan of procedure prolapse from the upper end of the bowel was prevented, and though it was true that prolapse might occur from the lower end, that rarely took place if the spur was kept at the lower end of the wound. The effect of this manœuvre when the bowel was opened was to leave the two apertures in the bowel of unequal size, the upper one admitted the index finger easily, but the lower one would only give passage to a good sized penholder. Besides this plan he also took care to suture firmly both ends of his original wound and did not utilize the whole of it for the attachment of intestine like Mr. Allingham. It was necessary to

make a large opening in the first instance so as to be sure of one's ground, but all but an inch or an inch and a half of the wound should be subsequently closed.

Mr. HARRISON CRIPPS, in reply, said he always closed the lower opening as well as he could. He usually put at least twenty or thirty sutures into the intestine, and this prevented the danger there was of the small gut being forced out beside the large bowel. As the bowel completely occupied the incision, he failed to see how the measure proposed by Mr. Jessett could be carried out. It was dangerous to fix the gut to an incision near the middle line. In one case in which he did this, internal strangulation resulted.

Mr. ALLINGHAM, in reply, said that the amount of gut he removed in his primary procedure was very trivial. In the supplementary operation he had removed as much as six or twelve inches. He always stitched the intestine carefully to the skin. He had found that the clamp was absolutely necessary to check the furious bleeding that was likely to happen when removing the gut in the supplementary operation.

November 2nd and December 14th, 1891.

THE PATHOLOGY OF INFLUENZA, WITH SPECIAL REFERENCE TO ITS NEUROTIC CHARACTER.

By JULIUS ALTHAUS, M.D.

MR. PRESIDENT AND GENTLEMEN,—I purpose to show in this communication that the virus of influenza attacks primarily, not, as is generally believed, the mucous membranes of the respiratory tract, but the nervous system of the sufferer, through the agency of the blood; and that the symptoms of the feverish attack, as well as the sequels and complications of the disease are owing to irritant poisoning of a definite portion of the nervous centres. The word “influenza” being somewhat long, and, as it appears to me, not very happily chosen, I have in the present paper frequently used the term “grip”—by which the disease is known in Germany and France, but spelt as an English word—as synonymous with influenza. I hope that this innovation may be generally accepted, not only because the term is short, but also because it graphically denotes the suddenness with which the disease attacks the patient. Another reason for accepting the term “grip” as equivalent to influenza is that it is really impossible to speak of the “influence

of influenza," as one often feels tempted to do when talking or writing about it; while the "influence of grip" may pass muster anywhere. There can be no doubt that the epidemics of grip of the years 1889-91 have been the most interesting medical event of late years, and that they have taught us a great many lessons which we did not know before. Indeed, the disease, not having appeared in England in an epidemic, or rather pandemic, form for many years past, was unfamiliar to the present generation of practitioners, more especially as in numerous cases the signs of catarrh of the respiratory organs, commonly called "influenza cold," and which were generally believed to be characteristic of the complaint, were either slight or completely absent, the most striking symptoms of the distemper having been in the majority of cases a sharp and short attack of fever, great physical and mental prostration, and severe pain in the head, body, and limbs, most of all of these symptoms ceasing as suddenly as they had appeared.

In accordance with our present views as to the mode in which infection takes place, I look upon the symptoms of influenza as owing to the action in the system of a special toxine secreted by a pathogenous bacillus. I regret to say that the results of numerous and laborious researches made by competent observers on the bacteria of grip flatly contradict each other, and that nothing definite is as yet known about the morphology of that micro-organism, or about the chemical constitution of its poisonous secretion.* All I feel it prudent to advance with regard to these points at present is that the life-duration of the bacillus seems to be in the majority of cases a short one, inasmuch as the stage of incubation of the malady does not appear to last longer than two or three days, and the attack of the disease from two to fifteen days, while the patient may remain a focus of infection to others for about a week or ten days longer. On the other hand, the virulence of the toxine secreted by the parasite appears to be most remarkable, causing, more especially when it falls on a suitable soil, an immense variety of severe symptoms, not only during the primary attack, but also in many cases for a long time subsequently, leading not unfrequently to a fatal issue, or to such destructive lesions of important organs as to tend to disable the patient for life.

* This gap in our knowledge has since then been filled up by the discovery of Pfeiffer's bacillus (January, 1892).

Why should the attack of grip end in some cases suddenly, with profuse perspiration and all the other symptoms of a crisis, leaving the patient weak, but really not much the worse for what he has gone through, while in other instances its course is much more protracted, and attended with dangerous complications and sequels? It appears to me that we can give a tolerably plausible answer to this question by reference to what takes place in pneumonia, which has been much better studied in this respect than grip. In pneumonia, as in most other infectious diseases, it is not so much the circulation in the blood of a special bacillus (Fraenkel's diplococcus), or the number of these micro-organisms, which kills, as the poison secreted by them. This poison, which has recently been isolated by Klemperer, causes the fever and the consolidation of one or several lobes of the lung, and endangers life by depressing the vital energy of the nervous centres of respiration and the heart's action. After this pneumo-toxine has circulated in the blood for a few days, an antidote to it—the anti-pneumo-toxine—is formed, by the aid of the poison itself, in the serum of the patient, and neutralises the poison so as to render the serum innocuous, when there is a crisis, and the patient recovers either completely or incompletely, according to the quantity of the anti-toxine which may have been formed.

Let us now apply the results of these researches to influenza, which appears justifiable on account of the similarity of the morbid processes in the two diseases. I assume, then, that the patient having acquired infection, a poisonous albuminoid secreted by a special pathogenous bacillus, and which I will call the *grippotoxine*, circulates in the blood, and causes the special symptoms of the feverish attack. In a day or two, however, an antidote, which I will call the *anti-grippotoxine*, is formed in the serum of the patient. Now let us suppose the quantity of this antidote to be in a given case sufficient for neutralising all the poison which is circulating in the blood; and there will be a crisis, with a sudden fall of temperature, profuse perspiration, and relief to the distressing subjective symptoms. If, however, the quantity of the antidote should be too small to neutralise all the toxine which may be present, the course of the disease will be protracted; there will only be a pseudo-crisis, and complications and sequels of different kinds will follow. By the aid of this theory we may also explain why *immunity* which has been acquired by a patient may subse-

quently be lost again. Let us suppose that all the anti-grippotoxine which has been formed in the serum has been lost, and that the patient is again exposed to infection. A second, or even a third attack of grip may then take place in the same individual, just as we meet occasionally with a second or third attack of measles, pneumonia, or rheumatic fever in the same patient. The theory which I have just proposed appears, therefore, to explain satisfactorily (1) why patients acquire influenza; (2) why they recover from it, either perfectly or imperfectly; and (3) why, after having had it once, they may contract it again a second or third time.

I now proceed to the next portion of my subject, which is to show that all the symptoms of the feverish attack of influenza are referable to irritant poisoning of a definite centre of the nervous system. Let me first show you the steps by which I have arrived at this opinion. Shortly after the visitation of influenza had commenced, I was surprised to see, both in hospital and private practice, a number of patients complaining of severe forms of neuralgia, loss of power, and a general break-up of the nervous system, which they attributed to an attack of grip which they had recently passed through. Some of these patients had been in perfect health before, so that the grip appeared to be the *fons et origo mali* altogether; while in others a neurotic pedigree or a previous syphilitic infection, or some other constitutional fault could be clearly traced, upon which the subsequent nervous affection had, as it were, been grafted. I also found that the number of nervous sequels which appeared after influenza was largely in excess of other post-febrile neuroses, of which I had seen numerous examples in the course of my practice. In comparing those nervous troubles which may be met with after such diseases as diphtheria, typhoid fever, scarlatina, smallpox, measles, erysipelas, and malaria with those seen after influenza, none of them—nor, indeed, all of them put together—approached in number the nervous sequels of grip. This I attributed to the circumstance that more than half the population of the country had lately been down with influenza, while the number of patients suffering at any one period from other fevers is always very much less. There was, however, also a much greater variety in the nature of post-grippal neuroses perceptible than in others, which run in comparatively narrow grooves. Indeed, it soon became evident that as a powerful

etiological factor of all kinds and forms of nerve disease influenza stands *facile princeps* among all infectious fevers. The only distemper which approaches grip in this particular quality is syphilis, which may also give rise to the symptoms of almost any nervous diseases. I find a still further analogy between these two infectious diseases in the circumstance that in both we may have a primary attack, secondary symptoms of a comparatively mild character soon afterwards, and tertiary affections of a more dangerous and obstinate nature, affecting the organic structure of tissues, at a more remote period. Grip also seems occasionally to revive an old syphilitic infection which has lain dormant in the system for years, and thus indirectly to give rise to certain diseases of the spinal cord, which are known to occur habitually on a syphilitic base. In comparing the degree of virulence of the two poisons, however, I have found that when the grippo-toxine attacks the structure of organs, it often does so with far greater ferocity and in a more ruthless manner than the syphilitic virus. Thus we see sometimes incurable blindness from optic atrophy, spastic spinal paralysis, and general paralysis of the insane becoming fully developed in a few days, weeks, or months from the outbreak of the feverish attack; while these diseases, when owing to syphilis, take years to become fully developed, and are also more amenable to treatment. When I had once realised this extraordinary tendency of grip to be followed by nervous sequels of almost any description, it was only a short step to the further inquiry whether the chief reason of this peculiarity might not be found in the circumstance that the distemper itself, in its primary manifestations, is not so much an infectious catarrhal fever, as has been generally assumed, as an infectious nervous fever. A clinical survey of the symptoms of the feverish attack rendered this, *primâ facie*, not unlikely, as many of them, such as headache, utter prostration of mental and bodily strength, delirium, coma, convulsions, &c., point unmistakably to the nervous system as their starting-point; while, on the other hand, catarrh of the mucous membranes and pneumonia have been completely absent in a large proportion of cases. Indeed, many patients have had influenza badly without having once coughed or sneezed.

The great varieties observed in the symptoms of the feverish attack of grip have induced a number of observers to assume three different forms of the disease, viz., the nervous, catarrhal, and

gastric variety. I wish, however, to impress upon you most strongly what I am convinced to be the fact, viz., that these three forms have not any different pathological characters, but that *influenza is always a true nervous fever*, the symptoms of which only differ as far as localisation of the grippo-toxine in different areas of the nervous system is concerned, and that the three forms just mentioned are perfectly arbitrary, however much sanctioned by authority. I shall, therefore, now endeavour to prove to you that the first or nervous form is that in which we have to do with the effects of the grippo-toxine upon the thermolytic, cardiac, and other centres of the medulla oblongata; that in the second or catarrhal form the special nervous mechanisms formed by the fifth pair and the vago-accessory nerves which supply the mucous membranes of the respiratory tract are suffering; and that in the third or gastric form the symptoms are owing to poisoning of those portions of the nuclei and branches of the pneumogastric nerves which supply the abdominal viscera, with occasional extension of the shock to the sphere of the splanchnic nerves, which constitute a vasomotor centre for the whole abdominal cavity, and which anastomose with the pneumogastric in the celiac plexus.

1. *Nervous Form of Influenza*.—One of the most important symptoms of this variety of grip is the fever, which shows considerable peculiarities. But before discussing this, let me ask the question, Are there any afebrile cases of influenza? Time will not allow me to enter further into this matter except to say that I have seen a number of cases of what I fully believe to be chronic infection of the nervous system with grippo-toxine, the principal symptom being intense mental depression, which may lead to suicide. Of the peculiarities of the fever of grip, I will in this place only state that there is no parallelism between the degree of temperature and the severity of the illness, as is the case in the eruptive fevers and other acute diseases, and that it is therefore unimportant from a prognostic point of view. How is the fever to be explained by the neurotic theory of grip? Most pathologists hold at the present day the doctrine that the fever heat is owing to increased production of heat, which is caused by irritation of the thermogenetic centre in the caudate nucleus. I have, however, long been of opinion that the opposite contention first put forward by Traube is nearer the truth, viz., that the fever heat is owing to increased retention of heat or diminished loss of it. This

theory has quite recently received considerable support from the experiments of Rosenthal, of Erlangen, which appear to have been very carefully made; and I would therefore explain the fever heat in grip by *congestion of the thermolytic centre* in the bulb, which regulates the loss of heat which is constantly taking place through the skin and lungs, and which includes for this purpose the vaso-constrictor centre which presides over the action of the blood vessels of the skin, the sudoriparous centre which controls the action of the sweat glands, and the respiratory centre which regulates the movements of the lungs (Hale White). As long as this thermolytic centre continues to be irritated by the grippo-toxine circulating in the blood, the fever continues; but as soon as so much antitoxine has been formed in the serum as is required for neutralising the action of the toxine, there is a crisis, with a sudden fall of temperature, profuse diaphoresis, loss of heat through the skin and lungs, tendency to sleep, and relief of all subjective symptoms.

I have just referred the fever of grip to congestion of the thermolytic centre in the bulb. Indeed, no one who has watched and considered the clinical symptoms of the feverish attack of grip will deny that congestion must be looked upon as the principal pathological process which is at work during that time in the system. It is true that the symptoms are often so severe as to indicate at first sight inflammation. More especially in children, grip begins sometimes with what looks like symptoms of meningitis. There is intense headache, vomiting, constipation, grinding of teeth, rigidity of the neck, convulsions, delirium, and coma; yet the sudden defervescence of the most alarming signs, which may be noticed twenty-four or thirty-six hours after their commencement, renders it certain that there can have been no inflammation or effusion, which require a much longer time for their resolution or absorption. Congestion, on the contrary, we know to be liable to very sudden modifications and variations under the influence of various agents, or after the cessation of certain causes. This opinion is also supported by the fact that in cases which looked like meningitis, a sudden improvement has often ensued in consequence of profuse epistaxis setting in. Moreover, we actually see congestion in that suffusion of the conjunctiva and swelling of the eyelids which are met with almost invariably in grip. Inflammation, however, does occasionally occur in various organs when the

irritation of the vaso-constrictor centre in the bulb reaches a very high degree. Thus we see meningitis (Fraser, Mackay), cerebral abscess (Bristowe), and a variety of other inflammatory affections, such as otitis, glossitis, keratitis, optic neuritis, endocarditis and myocarditis, nephritis, orchitis, &c. While I explain the occurrence of most of these inflammations by excessive irritation of the vaso-constrictor centre in the bulb, some of the more specific inflammations which are also liable to happen, such as erysipelas and pneumonia, in which special bacteria are known to be the exciting agents, are more probably owing to the loss of the power of phagocytosis in consequence of the illness. Microbes, which in health are habitually destroyed by the leucocytes as soon as they appear at the entrance gates of the system—*quærentes quem devorent*—are then readily admitted and allowed to multiply in the blood, with the result of causing specific inflammations.

The headache which, next to the fever, is the most common symptom of the nervous form of grip, is in the majority of instances owing to congestion of the membranes of the brain and of the sensitive portions of the cerebral substance, which is in its turn consequent upon irritation of the vaso-constrictor centre in the bulb. When the headache is particularly intense and prolonged, it may be due to inflammation of the parts just mentioned. In Bristowe's cases of cerebral abscess which I have already mentioned, constant and intense headache, which lasted throughout the illness, was one of the principal symptoms. In other cases it arises from catarrh of the frontal sinuses. The backache and pain in the body and the limbs, which are also very frequent, may be traced to congestion or inflammation of the spinal membranes and the sensitive portion of the cord, as well as of certain peripheral spinal nerves.

Delirium is frequent in the febrile stage of influenza, and sometimes consequent upon the headache where this is severe. The patient is then literally driven mad with pain. In other cases it is the chief symptom which overshadows all the other signs of the attack. The delirium and insomnia are owing to congestion of the cortex, whereby the grey matter is irritated; while somnolence and coma, which also occur rather frequently, must be referred to a more severe degree of congestion, whereby the brain matter is actually compressed. Where the congestion affects the central convolutions of the motor area of the cortex, we meet with

convulsions or paralysis, according to the more or less severe degree of the hyperæmia. Other symptoms of the feverish attack, such as subjective flashes of light, noises in the head, giddiness, deafness, loss of smell and taste, numbness of the head and face, &c., may also be accounted for by congestion of the different nerves involved, all of them, however, being in the last instance dependent upon the central event—congestion of the vaso-constrictor centre of the bulb.

Cardiac and respiratory crises, characterised by vertigo, fainting fits, syncope, dyspnœa, &c., which are not justified or explained by stethoscopic signs, point clearly to irritation and subsequent depression in the cardiac and respiratory centres in the bulb.

2. *Catarrhal form of Influenza*.—The peculiar feature of this variety is that we have, in addition to the fever, &c., symptoms of catarrh affecting more or less the whole extent of the mucous membrane of the respiratory tract. The grippal catarrh of this tract differs considerably from ordinary catarrh affecting the same membranes. In grip the inflammation extends to all parts, including the maxillary sinuses, the Eustachian tube, the tympanic cavity, &c., which does not occur in ordinary catarrh. In grip the serous or sero-sanguinolent flow from the naso-pharynx is more profuse. There may be also some or all of the symptoms of the nervous variety, with retching and vomiting, while the expression of the face denotes a greater degree of suffering than exists in ordinary catarrh.

The bronchitis of grip is also of a peculiar character, differing from ordinary bronchitis. It is generally very rapidly developed, and accompanied by a peculiar pain behind the sternum, and difficulty of breathing which is out of proportion to the physical signs. The cough is more distressing and obstinate, and the expectoration eventually more profuse than in ordinary bronchitis. These differences are also strikingly marked in the peculiar form of pneumonia which is apt to accompany or to follow grip. It is true that ordinary croupous pneumonia, as produced by Fraenkel's diplococcus, occurs in these cases, but this I believe to be only indirectly connected with grip. The system, being enfeebled by the invasion of an irritant poison, loses its power of resistance, and offers a favourable soil for the development of those bacteria which are always present in the fluids of the mouth, ready to invade the lungs when the entrance door is no longer barred to them. There

is, however, a form of broncho-pneumonia peculiar to grip in which it is not so much Fraenkel's diplococcus as the streptococcus pyogenes and the staphylococcus aureus which infest the sputum, and where the symptoms are of a much more insidious character. Want of time prevents me from fully entering into these differences, and I will therefore only say that in order to account for them we are driven to assume a different etiological factor for these peculiar forms of catarrh and broncho-pneumonia.

The nutrition and secretion of the upper portion of the air-passages are under the influence of the fifth pair of cranial nerves, experimental division of which causes keratitis, conjunctivitis, ulceration of the mouth, lips, tongue, and hard palate, and other symptoms. Irritation of this nerve, as caused by inflammation or the pressure of a tumour, gives, however, rise to a great many other symptoms, which I have fully described in a paper read before the Royal Medical and Chirurgical Society in 1868, and published in its Transactions for 1869. Amongst these symptoms are hypersecretion of conjunctival mucus, profuse catarrh of the Schneiderian membrane, leading to thick scabs filling up the nostrils, tendency to nasal hæmorrhage, hypersecretion of mucus in the mouth to such an extent that the patient is obliged to have a pocket-handkerchief constantly applied to it in order to prevent the liquid from running down the chin, while the lips appeared covered with froth, such as we see it in a patient who is in an epileptic fit. There is also ulceration of the tongue and mouth, and tendency to hæmorrhage from the gums. Irritation of the fifth nerve is therefore seen to give rise to catarrh of all the mucous membranes supplied by it; and I consider that the catarrh of the upper portion of the air-passages which we see in the catarrhal form of grip is owing to irritation of that nerve by the grippo-toxine. If time permitted, I should like to speak in connection with this, of other forms of inflammation, such as glossitis, parotitis, acute glaucoma, and more especially of that peculiar form of keratitis which is seen in grip. The observations of Galezowski and others on this latter inflammation show most plainly the neurotic origin of it, as its symptoms differ considerably from other forms of keratitis, and are almost identical with those described many years ago by Senftleben as occurring after section of the fifth nerve. The keratitis peculiar to grip has also been found to require an entirely different treatment from

that of other forms of keratitis. In the same way as the nutrition and secretion of the upper portion of the air-passages are under the influence of the fifth nerve, the nutrition and secretion of the lower portion of the tract, from the pharynx down to the lungs, are under the control of the vago-accessory nerves, which, like the fifth, have numerous anastomoses with the sympathetic throughout their course. The peculiar hacking cough which occurs in the catarrhal form of grip I would more especially ascribe to congestion or inflammation of the cough centre in the bulb, which is connected with the larynx by the superior laryngeal nerve.

There are few points in experimental physiology which have been so carefully studied, ever since the times of Valsalva and Morgagni down to the present day, as the lung affection following the section of both vago-accessory nerves. Suffice it to say that the consequence of this proceeding in all animals, birds alone excepted, is broncho-pneumonia. Paralysis of these nerves renders the glottis unable to close, and thus to separate the digestive from the respiratory tract; so that not only particles of food, but also, what is even more dangerous, the fluids of the mouth, which always contain pathogenous bacteria, enter the larynx and the lungs, and there act as excitors of inflammation. Death in three or four days is the inevitable result of the operation in most animals; but the actual cause of death is still somewhat doubtful. It cannot well be the broncho-pneumonia which kills, for birds die after the operation, although no lung affection occurs in them; and it seems most probable that death is owing to a variety of troubles caused by the removal of so important and complex a nervous mechanism. One of the principal of these troubles is gradual exhaustion of the respiratory centre in the bulb, as shown by a total change in the normal type of respiration. This exhaustion of the bulb is what actually occurs in the later stages of the broncho-pneumonia of grip when the patient is unable to rally. Birds die after vagotomy in eight or ten days with the symptoms of inanition, and the heart, liver, stomach, and muscles are then found to have undergone fatty degeneration, showing plainly the influence of the pneumogastric nerve on the nutrition of those parts.

Let us now consider at what part of the course of the fifth and pneumogastric nerves the irritant lesion produced in them by the grippo-toxine is situated. It seems to me evident that this lesion—whether congestive or inflammatory—must be very high up, as

the symptoms habitually implicate the whole extent of the area which is under the influence of these nerves. Moreover, their simultaneous affection which occurs in the catarrhal form of grip would lead us to assume a locality where they are lying close together. The two pairs of nerves are in closest contact, however, in the uppermost portion of the spinal cord and the bulb, where they originate with two nuclei, a small motor, and a large sensitive one, lying respectively on the top of the anterior and posterior grey cornua of the spinal cord. Irritation of this portion of the bulb is therefore shown to account for the symptoms observed in the catarrhal form of grip, just as irritation of certain other centres in the bulb has been seen to account for the symptoms of the nervous variety of grip. I have just mentioned that in my case of bilateral neuritis of the fifth nerve there was great tendency to hæmorrhage from the mucous membranes affected; and this observation leads me to consider another set of symptoms which is apt to occur in grip, and of which I have not yet spoken—viz., the hæmorrhages which may occur in various organs, such as epistaxis, bleeding from the gums, the external and middle ear, the stomach and bowel, the womb and the kidneys, &c. Such a widely spread hæmorrhagic tendency must have a central cause, and the observation I have just referred to shows the direct dependence of the same upon nerve irritation; so that I feel justified in contending that this tendency is owing to specific irritation of the vaso-constrictor centre in the bulb.

3. *Gastric form of Influenza.*—While in a large number of cases the digestive organs escape the morbid influence, instances are met with in which there is not only fever and headache, &c., but regular gastric crises, such as we see them in locomotor ataxy. The principal symptom is incessant vomiting, at first of food and mucus, and afterwards of serum, bile, and blood. This is accompanied with gastric uneasiness, cramp, and intense pain all over the abdomen. In other cases the bowel suffers severely, there being the symptoms of dysentery or choleraic diarrhoea. The gastric crises point unmistakably to severe irritation of the vomiting centre in the bulb, which is affected not only by stimulation of the central end of the vagus, but also by that of many afferent fibres in the same nerve. The pneumogastric nerve regulates not only the secretion of the gastric juice, but also the motility of the stomach, the glycogenic function of the liver, the action of the

pancreas, and even that of the intestines. On the other hand, the whole vascular area of the abdominal cavity is likewise under the influence of the splanchnic nerves, which unite with the phrenic and right pneumogastric to form the cœliac plexus, from which spring the phrenic, hepatic, splenic, mesenteric, renal, and other plexuses. It may thus be readily understood that a shock given to the nucleus of the vago-accessory nerve in the bulb may be transmitted to the whole of the abdominal organs by the nervous path just indicated, and cause more especially the intestinal crises of which I have just spoken.

Having thus traced the immense variety of symptoms occurring in the different forms of influenza to irritant poisoning of the bulb and the nerve nuclei contained in it, one other question remains for me to answer—viz., Why should the grippo-toxine tend to attack with preference the parts I have mentioned? To that question I can only reply by pointing to analogous facts, which have long been known, showing the existence of elective affinities of other poisons to other portions of the nervous system. Let me remind you that ergot of rye attacks with preference the posterior columns of the spinal cord, while lathyrus cicera lays hold in a similar manner of the lateral columns, and lead seeks out the anterior grey cornua of the same organ. With such striking instances before us, it may appear less surprising that the grippo-toxine should select for its point of attack another strictly circumscribed portion of the nervous system, the integrity of which we know to be of the first importance for the various phenomena of life.

RELATIONSHIP BETWEEN INFLUENZA AND THE NEUROSES.

By GEO. H. SAVAGE, M.D., F.R.C.P.

THOUGH in previous epidemics physicians have noticed the extreme nervous disturbance which may be associated with influenza, yet hitherto little has been written in England on the subject. Therefore having had considerable experience in the effects upon the nervous system produced by this disease, I have brought it before the Medical Society for discussion.

Many writers have recognised the relationship which may exist between fevers and neuroses ; but I shall be able to point out that the neuroses, of which we shall have to speak, may arise in cases in which the febrile disturbance has been very slight indeed, and in which the disorder did not seem to bear any direct relationship to the fever process. I shall have to give as my experience that, though the effects of the influenza are in many cases very similar, there is nothing which can be called special in the types or forms of mental disorder which may occur in connection with influenza.

The subject has been treated of by several physicians on the Continent, and I must refer to the volume recently published by Dr. Leledy, of Paris, on '*La Grippe et l'Aliénation Mentale*.' This book gives a very complete bibliography of the subject. Dr. Hack Tuke, in his recently published '*Dictionary of Psychological Medicine*,' gives a full account of the associated disorders as well as bibliography.

It is interesting to see that Arbuthnot noticed that, in 1732, there was a great run of hysteria, hypochondriasis, and other forms of nervous disorder following on influenza. Falconer, in 1803, described vertigo as a sequence of the disease ; while Stree-ter writes of delirium, affection of speech, and coma ; others, about the same time, described apoplectiform seizures and losses of consciousness. Dr. Handfield Jones, in his lectures on Functional Diseases of the Nervous System, published in the '*Medical Times and Gazette*,' calls attention to the variety of disorders which may depend on influenza. Sir Henry Holland wrote about two epidemics, which he had watched, in his '*Medical Notes*' (1832), and he recognised its probable air origin, and also its nervous associations. But I need not further occupy your time with authorities, having referred you to sources of information if you wish to trace the history of the recognition of the nervous symptoms of influenza.

I have made 55 cases the basis of my paper, though I have now nearly double that number to refer to. Of these 55, 33 occurred in men, and 22 in women. In 4 general paralysis of the insane developed after the influenza, and, in some degree at least, depended upon it as the exciting cause ; in 20 there were melancholic symptoms ; and I must say that my general impression and that of many of my friends was, that the great majority of post influenzal psychoses were melancholic in their form ; but

when I came to the fixed data derived from various asylums, I found more cases suffering from other forms of insanity than I expected. In 13 of my 55 cases there was acute mania of the ordinary type; in 5 delirious mania; in 6 delusional insanity; and in 7 more or less marked mental weakness.

As far as my present returns allow me to judge, 70 per cent. recovered, while three of my cases have died. These are but provisional returns, and it will be long before I can give really valuable evidence as to the prognosis; suffice it to say, that my own impression is that the majority of cases recover, unless the neurosis is an expression of marked decay of the nervous system, resulting from age or general paralysis of the insane.

I have to thank Drs. Wigglesworth of Rainhill, Mickley of St. Luke's, Rees-Philipps and Caldecott of St. Anne's Sanatorium, Virginia Water, and Dr. Bonville Fox, for their kind assistance in giving me returns from the patients under their care.

I shall submit the notes of a few cases and sum up, as I find the time at my disposal permits.

Influenza.—Change of Temperament.—Acute Disease.—Mania.

Mr. —, seen October 12th, 1891, aged 21, butcher, mother's brother insane; father's brother also insane; in both cases some special trouble or injury considered by the friends to be the cause of the insanity. This patient has been very hardworking, and remarkably strong and well built. He has not been very sober, but never was actually drunk, or neglected his work; he usually attended early market. He had influenza about four months ago, and was rather ill with it, having some lung complication (one brother died of phthisis after measles). He got over the influenza, but soon after it was noticed that he had changed in his manner, so that he was irritable and even rude without occasion, and would suddenly turn round and say to his mother or sister, "what are you talking about," as if he thought someone was referring to him. He did not sleep very well either. He contracted syphilis about a month or six weeks since, it is not quite clear when. He does not seem to have been treated for the disease, and this may have something to do with his manner. About eight days ago he consulted a doctor, who found him with sore throat and rupial patches about the limbs and body; he was thin and ill-looking; he had a temperature of 102° ; he complained of little or no pain; he was dull rather than depressed. He rapidly got worse, so that, though the temperature fell, yet he was in a semi-delirious state, and to-day has been shouting for hours together, and not taking food or medicine.

I found him prowling about his room in a dressing-gown; he was in no way violent, but rather preoccupied and silly; he allowed me to examine him; he put out his tongue which was fairly clean and moist; pulse rather over 100; nothing noteworthy; no lung trouble to be found on rough examination; no albumen in urine; he was thin and weak.

A case of acute mania with acute syphilis. He may be tried with

mercurial inunctions at home if he will take food, otherwise he must go to Bethlehem. He was sent to Bethlehem, where he continued maniacal till January, 1892, when he began steadily to improve, and was discharged cured.

Acute Delirious Mania after Childbirth. (? Influenza as a Cause.)

Mrs. —, married eleven months, of fairly healthy stock, but always a little nervous ; she had a natural labour after a natural pregnancy (her first) ; she went on very well for four days, then, without cause, she had a severe headache. Temperature went up to 104°, and the friends and the medical man became alarmed. There had been a great deal of influenza about, and the medical man diagnosed this as the cause.

When I saw the patient she was suffering from acute mania of the delirious type, and was very dangerously ill.

She was treated very freely with stimulants, and the only time there was a relapse and fresh danger was when these were for a time withdrawn ; after a period of slow convalescence, marked by great physical and mental depression, she got quite well.

Exaggerated Influenza.—Depression.—Tendency to Hallucinations.

Mr. D—, theological student, single, aged 24, was formerly a farmer, but afterwards went to college, and there was in good health (sister melancholic). A little more than two years ago he felt depressed, and then had a rather severe attack of influenza. He got over this, but has been dull and wretched ever since ; he has no pains, but is nervous, fancying he is watched ; he also thought people spoke about him. He had no visions or bad smells. He had been a masturbator, but not much nor of late. He had been very sober, and no great smoker ; he did not sleep well ; he falls off to sleep, then wakes early, and cannot sleep again. He has not been suicidal, though feeling the uselessness of life ; he tried working very hard on the land like a common labourer ; for a time was better, then relapsed ; was without energy, could not get up in the morning, had no heart to read or play ; he has varied a good deal, being brighter for a time, then worse again. Now he feels that he cannot do what he wants, and he is also nervous that people are noticing him. He has neither hallucination of hearing nor of sight ; he has no bad taste or smell ; the circulation is fair. His appetite is not good, his bowels are confined, and he feels stronger than he appears.

This seems to be a case of mental depression which was accentuated by an attack of influenza.

As a voluntary boarder at Bethlehem Hospital he has been discharged cured.

Post-Influenzal Melancholia.

Mr. H—, seen July 19, 1891, engineer, married, no children ; father died of age and rheumatic cardiac complications ; mother died of "general break-down" at a great age ; she also was depressed before then ; and this seems to be rather the way with his aged relations. He has lost several brothers and sisters of phthisis. He has been a very healthy man, never having had any serious illness of any kind ; he has been very sober in every respect ; he has been successful, but has had an enormous amount

of work of one kind or another, and has not taken much rest. Early in May he got a feverish cold, which his doctor has no doubt was influenza ; then he was very weak after it, and depressed, and he went away for a time by medical advice, but did not get better. He next saw a physician, who suggested his going away for a month ; he did this, but was no better. Then he was ordered to go to Switzerland ; he was preparing, when he became much worse, and his mind was noticed to be affected. Mental symptoms had lasted ten days, when I was asked to see him. He was in bed ; he had a very distracted look ; he would not listen to me, but went on in a rambling way about his "being altogether a fraud," that he was ruined, and that his wife would come to beggary ; he was ever repeating the same complaints of the evil he had done ; he accused himself of embezzling and robbing. His skin was moist and normal in temperature, pulse rather rapid, 90, weak, not hard ; pupils rather smaller than normal, equal, discs not to be seen ; he has no pain anywhere that he complains of ; he has had several rather severe griping fits to-day, as if from flatus ; his urine is high coloured, and there has been some albumen in it ; his tongue is thickly coated with a patchy milky-white fur.

I think this is post-influenzal melancholia. He ought to have quinine, some stimulants and hypnotics at night, as he sleeps badly. Paraldehyde has done fairly of late. I ordered chloralamide, great care as to suicidal tendencies, and as to feeding. He ought to get over the attack, if he takes food enough, but there is some danger. In any case, he must be kept quiet for three months, and ought to try a voyage after his recovery. After great mental improvement he had a relapse, but by January, 1892, appeared to be well, and has remained so.

Influenza in a Depressed Patient followed by Epileptoid Fits and Marked Melancholia.

Mrs. O——, seen October, 1891, married, 57, with healthy adult children, youngest 19. Has been a strictly sober, active-minded, religious woman, who has always been anxious-minded about her duties ; she has never been robust, but healthy, never having had any serious illness, no gout or rheumatic troubles ; she passed the menopause easily about eleven years since, but has been more anxious-minded ever since ; she had very bad digestion, was very thin ; she did not mind this, but consulted, at request of husband, many doctors. About two years ago, Playfair had her for seven weeks under treatment (Weir Mitchell), she gained flesh and seemed brighter in all ways, the constipation from which she suffered being less. She went last spring to the south of France, and she got influenza about the end of March, not severely ; she did not take much care of herself ; she felt depressed. She went about the end of April away for a tour, and one day at lunch horrified those with her by turning purple, thrusting things into her mouth and then falling. She was got to bed, and after "sleep" for a time she got up and wanted at once to resume her journey, not recognising that anything had happened. She was kept in bed, and in a day was well enough to go on. From that time she was more worrying and depressed, anxious about everything and everyone. She varied as to sleep and food. She got a little better for change to seaside, but soon fell back again. She, about three weeks ago, began to trouble more and more about the state of her soul, and could talk of nothing else. She said she felt that she had all her life professed to be a christain, but never really was ; that she had been a hypocrite, and that knowing what was right, she had done wrong ; she had blasphemous and

horrid thoughts passing through her mind, and could not check them; she only wished to be sent to an asylum. She became very excited for a time, and unfortunately got a second attack of influenza and died of pneumonia, being quiet and reasonable at time of death.

Post-Influenzal Automatism with Homicidal Ideas.

Mr. J——, seen October 9, 1891, married, with three healthy children. Employed in the Excise, where he has been for over twenty years without a single complaint for misconduct or the like. He has always been promoted on the earliest possible occasions. He has been very temperate in all ways. His father died of "atrophy" (he thinks phthisis); mother living. Twelve years ago had a rather severe head injury, but has felt no ill effects from it. End of June, had influenza with severe headache, high temperature, passed very little water. He was laid up for two weeks; went to work feeling weak; he worked all right for two days, then he says he lost all consciousness of what he was doing, though he did his work accurately and without any complaint, the work being such as needed special adaptation to circumstances. He then came to himself in a depressed state of mind, was very weak, and felt that he *must kill his youngest child*. He had been sleeping badly. He only remembers leaving home, and from time to time he recalls being at certain towns, such as Lewes. He went 100 miles on foot, wearing his boots to pieces. He then went to Bow Street, where he gave himself up for killing his child. He cannot tell whether the last clear idea on his mind being to kill the child, or the dread of doing this was the cause of his leaving home. Police surgeon said he was suffering from delirium tremens. He was sent with female attendant to Brighton. He was looked upon as reprehensible and degraded. He got better, had no more return of infanticidal ideas. He was sent to work in Sussex. He was much better when working in the open, but still very nervous, and not sleeping well. He now complains of confusion, inability to do his calculations; he has giddiness and some constant pain in left occipital region. Pupils wide, sluggish, discs normal, rather pale, knee reflexes exaggerated, appetite fair; he looks strong. I think this is post-influenzal, and though he probably will get well, there is grave cause for anxiety for some months. He ought to be off all work. There was no ground for the diagnosis of delirium tremens.

Drink.—Menopause.—Influenza.—Mania.

Mrs. E——, married, 51, still menstruating, though the periods are variable in time and degree. She has been a drinker all her life, or for many years anyway; beer has been her chief failing, but anything was taken that came in her way. Her father died of some form of mental disease in an asylum after nine years, there is some slight evidence that it might have been general paralysis. Her brothers have been scapegraces more or less. Up to May, she had been drinking; she never had delirium tremens. Her husband had influenza on May 7th, 8th, and 9th. She nursed him day and night without sleep, and she herself started with influenza on May 11th. Her temperature was high up to May 13th; she was quite rational but excited, but by May 19th she was wildly maniacal, and was brought to her home from the sea-side where they were on May 27th, in that condition. She was not seen by the doctor till June 11th. Her temperature was then 100°; it has never been above that since, and by June 30th was normal and remained so. Pulse about 100. When ill

with influenza she had no stimulant, but after a time a doctor put her on a quantity which had to be knocked off on return home. Now she has *not the least desire for drink*. She slept badly for a long time. At present she varies a good deal, sleeping with or without draughts, then for a night or two not sleeping whether she have chloral or not. A summary of her condition since then is as follows:—Enraged at sight of husband whom she mocks, assaults, and abuses. Uses foul language, and at times is obscene or low in talk, swears, has been coarse in eating, but now she is all right; has been untidy, now is simply negligent; has no affection for those near her. Her appetite is good, bowels a little confined, urine clear and normal; she has a very congested alcoholic aspect. The causes of trouble are alcohol, hereditary taint, climacteric and influenza. The prospect is fair in time. Perhaps a year may elapse before improvement ensues, and then in a quiet home, not her own.

January, 1892.—Patient was placed under special care in November, and has slowly improved, and is now quiet and contented, and there are no signs of mental deterioration, still no return of dipsomania. She ultimately recovered and returned home.

Nervous Weakness.—Influenza.—Fits.—General Paralysis.

Mr. T—, seen with Dr. Delepine and a friend at Thornton Heath, married, four children, mother imbecile, father died of some spinal disease, with mental affection in the end. Has been in Post Office for years, very steady, not over-worked in any way, a total abstainer, non-smoker, and a vegetarian. Has had good health, but has not been robust; has amused himself with flute playing, walking, &c. His wife says, “for four years he has not been himself, that he had sciatica for years, but he did not take opium or other things for it, the sciatica was one-sided.” He had influenza about Whitsuntide; this was not very severe, but a few weeks after he had a series of very severe apoplectiform fits, twelve in number, rapidly following each other, he had right side affection and aphasia; he recovered. He never had fits before. Since this he has been unfit for work though he has tried it. His memory is failing, his writing getting very bad, and his flute playing is defective, chiefly in time, yet this is not recognised by him. He also has changed in habits; he buys useless things; thus he would buy a big cake in the morning, and in the afternoon would buy another; he was reckless in going by train with or without tickets; he has got into trouble by stealing flowers and other things, he is rather sleepy, he eats fairly, and will not willingly see a doctor. I went in with a friend of his without any name being given; he did not object, he went for his flute but did not play, he was in a monosyllabic state. He had small pupils, speech very slightly hesitant but slow; there was loss of expression. He did not know where his family had been for holiday; he did not remember any recent things, but I could not get him to converse.

I believe he has early general paralysis, and ought not to be at large, or he will get into trouble.

In hands of police at Gravesend in February, 1891, to go to Bethlehem Hospital. Was kept in Bethlehem for three months, and thence sent to County Asylum as an undoubted case of general paralysis.

General Paralysis following on Influenza, in a Young Man.

Mr. D. C—, seen October 16th, 1891, single, aged 22, steady, very active and healthy; he had influenza with a high temperature, he neglected

it and trusted in cold water. While ill he seemed sad and uncertain, changed in his ways, and so was sent for a holiday on to the Continent. He became extravagant and wasteful of money, and had to be brought home. I found him sitting in his room in a thoroughly weak-minded state; he was expressionless, his skin sallow and greasy, his facial muscles tremulous, he replied to questions very slowly; pupils dilated, sluggish, lifeless; gait uncertain; writing shaky. I have very little doubt he is suffering from general paralysis. Considerable physical improvement and some mental gain ensued. Doubtless this is only a remission. The remission has continued, making the diagnosis doubtful.

Syphilis.—Influenza.—General Paralysis.—Theft.

H. —, married, aged 35, three children, sober. Twelve years since contracted syphilis, but had few constitutional symptoms. Had cramp four or five years ago and some ataxic symptoms. Six months ago had influenza, and since then has steadily lost ground in all ways. He had progressive loss of memory, followed by buoyancy and extravagance. He made serious mistakes in business, and at the German Exhibition stole things, or rather put useless things in his pocket. I found him weak, expressionless, and tremulous; he was very buoyant and extravagant, his speech was hesitant, and his writing shaky. His knee reflexes deficient. Pupils large and unequal, optic discs vascular. He was sent to a quiet village in the north, but soon became beyond control and was sent to an asylum.

Cases reported by Dr. Bonville Fox, of Brislington House, Bristol.

(1.) A clergyman, married, aged 43, childless but in good circumstances, was admitted February, 1890, with insanity of two months' duration. This was the first attack, and was preceded by and attributed to a slight attack of influenza. He was melancholic on admission, the depression becoming more profound. He was for a long time depressed, but, after being sent to the seaside, improvement ensued, and he was discharged recovered January, 1891. In this case there was very strong hereditary predisposition. He also had been given to self-abuse for many years.

(2.) The wife of a clergyman, age 29, with several children, has had two or three previous attacks of mania. The present attack has been similar to the others, and has lasted four months. She had influenza a short time before this breakdown, and there was also anxiety about the death of a friend. She was sent out convalescent after about four months' treatment. Recovered and kept well.

(3.) A married lady, aged 24, was admitted in August, 1890. She had recently had influenza, and her symptoms of insanity was of four days' duration. In this case two previous attacks of insanity had occurred at the ages of 14 and 22. The progress of the case was slow, but she improved, and was ultimately removed to another asylum.

Cases from Raxhill Asylum, contributed by Dr. Wiglesworth.

(1.) Mary A—, married, 57, formerly intemperate; influenza fourteen days before admission; kept up four days, then in bed two days, mental symptoms nine days before admission, after that kept in bed.

Acutely delirious before admission for four days ; on admission suffering from acute delirious mania, catarrhal pneumonia of both lungs, more extensive on the left ; was fed by nose for six days. She was insane for fifteen days, then slowly recovered, and was discharged in three and a half months.

(2.) Mary D——, married, 38 ; influenza eight weeks before admission, bronchitis present, head symptoms came on three weeks later, she was subacutely maniacal on admission, her bodily condition being fair. She got better, but is a chronic drunkard. Her stay in the asylum was three and a half months.

(3.) Charlotte L——, married, 55, temperate ; influenza ten weeks before admission, no complications ; she had nursed husband through an attack of typhoid ; after his recovery she grew dull, would not take her food well for five weeks before admission. She was very melancholic, and in weak bodily health. In her case there was strong heredity. Influenza did not play a very clear part in the causation, but yet was credited with partial causation. No improvement mentally by January, 1892.

(4.) Margaret G——, single, 55, temperate ; had influenza in the spring (six months before report) ; never got over it rightly. Mental symptoms about one month later. On admission she was depressed and melancholic, being actively suicidal. Physically thin and weak, mitral stenosis with regurgitation ; this is associated with renal disease and old pleurisy. Improved greatly by January, 1892.

(5.) Catherine A——, married six weeks, aged 18 ; influenza two weeks since, was in bed fourteen days, grew strange the very day she got up, when she was still very weak ; rapidly developed acute mania. She had a strong hereditary taint. She recovered.

Case of the General Paralysis following Influenza.

Thomas E——, married, 35, grocer ; admitted June, 1891, suffering from acute mania of the general paralysis type. Talking incessantly very incoherent, dirty in habits. He remained in this state till time of death, September, 1891. He had influenza three weeks before admission, and appeared to recover, and returned to work for two or three days when the mental disorder became manifest.

Case of Previous Attack.—Drink.—Influenza.

Richard Mac——, single, 20, labourer ; admitted June, 1891. He was in Prestwich Asylum in 1888. Said to have been mesmerised by friends, and left helpless in bed. He then recovered, and has been well until he got influenza, with which he was laid up for a week. He then returned to work for a week when mental symptoms appeared. He had been drinking about the same time, but it is not known to what extent. When admitted he was suffering from melancholia, passing into acute mania, which continued. He had hallucinations of hearing, and fancied that people knew his thoughts. In January, 1892, still maniacal.

Case of Senile Dementia.

James P——, aged 70, widower symptoms of two months' duration following influenza. This case was incurable. Died.

Post-influenzal Insanity. Holloway Sanatorium.

Initials.	Sex.	Age.	Date onset of influenza.	Date onset of insanity.	Date of admission.	Form of insanity.	Course of disorder.	Result.	Remarks.
A. N. ...	Female	38	June, 1891	June 28, 1891	Aug. 7, 1891	Acute mania with delirium	High temp.; tremor; dry tongue; delirium; hallucinations; vivid Ordinary course; hysterical; flip-pant; emotional; variable	Still under treatment; rapidly improving; memory of past very hazy Recovered	Will probably be quite recovered in from 6 to 10 weeks.
A. F. ...	Do.	30	May 26, 1891	June 2, 1891	July 13, 1891	Acute mania	Excitable; emotional; variable; loquacious Ordinary course	Sept. 28, 1891, still under treatment; rapidly improving Still under treatment; improving Died May 24, 1891	This is her 2nd attack, the 1st attack having occurred 4 years ago.
M. S. ...	Do.	47	June 14, 1891	June 20, 1891	July 3, 1891	Hypochondriasis with delusions of pregnancy Acute mania	Typhoid symptoms very marked, e.g., T. 104-106°. Tongue, &c., black; tremors; all food refused; fed every four hours with tube night and day; subsultus tendinum; violent delirium and restlessness		P.M. May 26, 1891. Very similar to those in succeeding case. Will give full details of both cases if desired.
A. L. C. ...	Do.	26	April 25, 1891	May 8, 1891	June 10, 1891	Acute delirious melancholia			
M. A. D....	Do.	45	May 5, 1891	May 10, 1891	May 14, 1891				
A. H. H....	Do.	34	March, 1890	About June, 1890	May 14, 1891	Acute delirious melancholia	Typhoid symptoms very marked; T. 102-104°; subsultus; black tongue, mouth, &c.; all food refused; fed with tube every 4 hours night and day	Died May 22, 1891	P.M. May 25, 1891. B.M. Membranes and cortex acutely congested all over, but especially marked at fissures of Sylvius; milky patches following ditto: cortex pale; brain substance tough and anæmic; no excess of serum; <i>stomach</i> lined with slimy yellow mucus $\frac{1}{8}$ -inch thick.

Post-influenzal Insanity. Holloway Sanatorium.

Initials.	Sex.	Age.	Date onset of influenza.	Date onset of insanity.	Date of admission.	Form of insanity.	Course of disorder.	Result.	Remarks.
L. B. O. ...	Female	26	About 10 Feb., 1890	Feb. 1890	Nov. 3, 1890	Active melancholia. Suicidal. Unpaid dinner	Ordinary course	Recovered April 17, 1891	Remained in perfect health since discharge up to present date.
M. L. ...	Do.	54	About 14 Mar., 1890	Mar. 20, 1890	April 24, 1890, transf. from Peckham House	Hallucinations of all senses. Delusions of persecution, especially of corosexual type	Abusive; violent; filthy in conduct and language	Transferred to private care; "relieved" April 16, 1891, but very slightly improved mentally.	
G. W. ...	Do.	36	Feb. 20, 1890	Mar. 3, 1890	Mar. 13, 1890	Acute mania	Steady progress to recovery	...	This was a second attack of insanity
G. E. O. ...	Do.	41	Oct. 27, 1889	Nov. 1889	Private asylum first; St. Anne's, Jan. 1890	" "	Steady progress; recovered Oct. 1890	...	Mania followed directly on influenza, which occurred after childbirth.

Private Cases.

Initials and sex.	Age.	Date and duration of influenza.	Approximate date of onset of insanity.	When first seen.	Form of insanity.	Progress of case.	Remarks.
Mrs. E. ...	51	May 11, 1891	May 18, 1891	Under treatment May 27 Oct., 1891	Acute mania	Slow progress	Intemperate; father insane at time of case.
Mr. N., married.	35	April, 1891	Almost at once		General excitement of G. P.; stealing	Steady degeneration	Syphilis 12 years ago; loss-motor ataxy 4 to 5 years.
Mr. C., single ...	22	Aug., 1891	Sept., 1891	Oct., 1891	Excitement passing into dementia	Some improvement	Heridity; exposure during influenza.
Mr. W., " ...	21	July, 1891	July or August, 1891	Oct., 1891	Change in temper; mania	Deliriously maniacal; weak; then recovery, January, 1892	Heridity; acute syphilis; drink; pneumonic complications.
Mr. L., " ...	26	Early in 1891	Some months after	Oct., 1891	Hysterical	Violent mania	Severe attack of influenza; lost consciousness at onset.
Mr. J., married.	48	June 1891; two weeks	Directly after	Oct., 1891	Partial dementia	Slow progress to recovery	Heridity; severe attack of influenza.
Mrs. O., " .	42	Spring, 1891	Six weeks later	Sept., 1891	Melancholia; restless	Slow progress to recovery	Spasmodic asthma; relieved during insanity.
Miss B., single ...	27	Dec. 26, 1890; relapse	Followed relapse at once	Sept., 1891	Hysterical; delirious	Steady progress to recovery	Neurasthenic in character.
Miss M., " ...	27	Jan., 1891, influenza and a relapse	Followed relapse	Sept., 1891	Neuralgic; sleepless, and weak of will	Slow and variable improvement	Pneumonia; previous attack of melancholia.
Mr. T., married.	42	May, 1891; relapse	May; after relapse	Aug., 1891	Fits; dementia, G. P.	Steady degeneration.	Neurotic stock; great worries.
Mr. F., widower.	72	May, 1891; relapse	End of June	July, 1891	Sleepless; melancholia	Quite well in September	Was simply depressed before influenza.
Miss G., single...	37	May	Gradually after	July, 1891	Sleepless; irritable and depressed	Improved steadily but slowly	
Mr. W., married	40	Jan., 1890	Delusions followed at once	July, 1891	Irritable and suspicious	No real change	
Mrs. T., " "	34	May, 1891	At once	July, 1891	Sleepless; melancholy with stupor		Had "brain fever" 10 years ago.
Miss M., single...	60	June, 1891	Never right since	July, 1891	Sleepless; partial dementia.	Apparently becoming weakminded	An overworked man.
Mr. H., married	50	May, 1891	Depressed at once	July, 1891	Variable; depressed, and hopeful	Slow progress; recovery	
Mr. H., " "	34	May 23, 1891	June 8; maniacal	July, 1891	Became suspicious and deluded	Steady progress; well in September	Previous attack of insanity; family neurotic.

Initials and sex.	Age.	Date and duration of influenza.	Approximate date of onset of insanity.	When first seen.	Form of insanity.	Progress of case.	Remarks.
Mr. C., single ...	35	Feb. and March, 1891	July, 1891	July, 1891	Was peculiar and suspicious, but managed to do his work till he had influenza; since then he has become a typical and incurable case of delusional insanity		
Miss F., "	31	May	At once changed in manner	July, 1891	Restless; maniacal	Sent to asylum; recovered September	Neurotic family.
Miss H., "	30	Aug., 1890	Never right after	July, 1891	Hallucinated and hysterical	Sent to asylum; has chronic look	
Mr. S., married...	49	May, 1891	Food; depressed at once	June, 1891	Sleepless; loss of attention; hypochondriasis	Slow improvement	Previous illnesses due to other causes.
Miss D., single ...	52	May, 1891	"Soon after"	June, 1891	Delirious mania	Sent to asylum; recovered September	Weakness from nursing mother.
Mrs. W., married	23	Feb., 1891	From the time of influenza developed obsession of ideas and hypochondriasis	Improved only to a small degree	Peculiar before influenza; heredity.
Miss G., single ...	28	Dec., 20, 1891	In a few days	Jan. 7, 1892	Sleepless; hysterical mania	Improving	Mother insane.
Mr. L., "	24	1890	Shortly after	Nov., 1891	Dull; apathetic; delusions	Voluntary boarder in asylum; slow improvement	Influenza accentuated previous depression.
Mrs. M., married	59	Feb., 1891	Shortly after	Nov., 1891	Sleepless; dull; miserable	Slow improvement	Depressed slightly before influenza.
Mr. M., single ...	25	Jan., 1892	Jan., 1892	Jan., 1892	Sleepless; restless; suspicious; maniacal		

St. Luke's Hospital.

Initials and sex.	Age.	Date and duration of influenza.	Approximate date of onset of insanity.	Date of admission.	Form of insanity, &c.	Progress of case.	Remarks.
In 1891. J. A., male	61	In June; a fortnight	Sept. 6, 1891, though weak ever since influenza	1891. Sept. 11	<i>Melancholia</i> .—Constantly praying aloud for forgiveness. Highly emotional. Imagines some one wishes to do away with him	Has improved considerably, but is childish, and generally weak physically and mentally. Still emotional. Jan., 1892. General senile decay. <i>Still in hospital</i>	Had a good deal of business worry and anxiety previous to attack of influenza, but showed no signs of mental defect.
S. S., "	39	End of May, lasting nearly three weeks	During first week in July, about previous to admission	July 27	<i>Mania</i> .—As a rule excited, sometimes depressed. Refused food	No improvement in mental condition. Died, Aug. 9, 1891, of uremia	Patient had masturbated freely, and there is reason to think had been desponding about two years.
F. F., "	23	Early in June, lasting a few days	A fortnight later	July 2	<i>Mania</i> .—Excited, rambling and incoherent	No signs of improvement whatever—worse if anything. Jan., 1892. No gain. <i>Still in hospital</i>	Had had some business worries previous to June, though not serious. Not hereditary. Masturbator.
Edith K., female	26	About middle of May —date not stated	May 22 (about)	June 6	<i>Melancholia</i> .—Very weak when admitted. Refused food. Believed herself very wicked	Improved considerably; was <i>transferred to Bethlehem</i> , Sept. 26	Always emotional and religious. Had been a good deal mixed up with Salvation Army.
T. B., male	54	In May, latter end; duration not stated	Prostrated and depressed from date of influenza	Aug. 8	<i>Melancholia</i> .—Believed he was lost. Refused food. Very depressed	No change for a month, then slow, and, latterly, very rapid improvement. Discharged well	Had had a good deal of business worry previous to influenza.
In 1890. S. A. R., female	35	Feb. 20 to 25, 1890	Noticed on March 1	1890. Mar. 13	<i>Melancholia</i> .—Much depressed; thought she was dead. Difficult to get her to take food	Improvement very slow. Always depressed and sulky. An unsatisfactory case. Jan., 1892. No real gain	<i>Discharged, Sept. 18, 1891</i> , at request of friends. <i>Not recovered</i> .
T. D., female	66	In Jan., 1890. Exact date not known	Almost directly after influenza	June 7	<i>Melancholia</i> .—Depressed. Refused food. Believed she was deserted by husband, and was afraid that attendants and patients were going to leave her. Worried a great deal	Improved slowly at first, then considerably, both physically and mentally. <i>Discharged</i> after trial (informally). Husband refused to report on her condition	Had an attack of melancholia nine years previously, and it is doubtful whether she had ever quite recovered from it.

RELATIONSHIP OF INFLUENZA TO THE NEUROSES.

Whatever the origin of influenza, there is no doubt that the nervous system is seriously affected during the process, and may suffer after effects. In former epidemics writers have described the occurrence of hysteria, hypochondria, and various other forms of mental disorder, generally of a low or depressed type. Several writers have more recently considered the after effects of the recent epidemics, but this has not yet been done in England. Though many independent writers have reported instances, they do not claim any originality in describing neuroses as following upon influenza. There is, however, sufficient interest in the cases to justify my putting together my experience.

Influenza seems to attack the sane more than the insane population of asylums. The statistics of Morningside, Edinburgh, have shown this well, but Dr. Sisley has shown that the same holds good of prisons, so that it really seems to depend on their restricted liberty and their reduced danger of contagion than on their nervous state.

It was marked by various neurotic symptoms, such as neuralgic pains in limbs, &c., in the nervously weak and the strong, but the true neuroses rarely appeared except in persons who were by heredity, previous attacks, or other causes, such as constitutional syphilis, in a state of nervous instability.

The after effects had no distinct relation to the severity of the influenza. The period at which the symptoms developed varied very considerably, so that in many cases it is hard to be quite sure as to the casual relationship between the two, and in some cases it is only by inference that one can give influenza as the cause, but I think that if in a certain number of cases a direct relationship can be traced between the influenza and the nervous symptoms, and if in certain other patients previously healthy, but who had influenza, a similar train of nervous symptoms follow, though after a period of apparent recovery, one is justified in saying that there may be a casual connection between the two, more especially as influenza is so very prone to relapse or recrudescence.

The occurrence of previous attacks of insanity, the existence of alcoholism, the presence of some cause of degeneration, such as syphilis or the menopause, the presence of neurotic inheritance, or

of old brain injury, are almost necessary for the development of insanity after influenza. Any variety or form of neurosis may occur, from the mildest hysteria to general paralysis of the insane.

Nervous symptoms of all sorts may occur quite independently of insanity, so that there may be delirium of considerable gravity with loss of consciousness during the acute period of the disease. Simple neurosis of various kinds may occur with and after the attack, of these the most common are sleeplessness, amnesia, more or less general neurasthenia, hypochondriacal and self-conscious feelings, neuralgia; rarely fits of an epileptic nature and diabetes have been reported as sequelæ. Of the more developed nervous disorders I have had examples of delirious mania of a severe and even fatal type, acute mania of a simple and hysterical form, hypochondriacal melancholia and other forms of melancholia; the self-conscious state has in some cases grown into true delusional insanity with hallucinations of the senses: in one case a strange form of automatism appeared, and in this case and in one other crime might have resulted, so that there is a medico-legal aspect to the subject as well as a medical one. In several cases general paralysis of the insane has followed directly on the influenza; in most of these cases there has been evidence that there were signs of degeneration before the onset of the influenza, but after this the disease was discovered. There seems to be great differences in the frequency with which severe nervous symptoms develop, for whereas in some cases medical men have attended several hundred cases of influenza without a single case of insanity occurring, others have had quite a considerable percentage. I cannot discover that the method of treating the influenza had any influence on the production of the insane disorder. Patients who, as a result of influenza, become insane, as a rule recover. There are two classes in which there is great danger, those in which acute delirious mania occurs, and in those in which general paralytic disease is present, some of the older patients who have begun to lose vigour, run great risk of becoming permanently insane. The course of the disorder is marked by frequent relapses, and is often tedious.

I have found that attention to the general condition is of the most importance. Thus for a time isolation, with watching is needful, for undoubtedly many suicides have resulted during the depression following influenza. Good food is advisable and sea

air, with restfulness rather than travelling. I have given the various hypnotics, but find morphia in the older cases, and sulphonal in the rest, the most efficacious.

To complete the subject, I must add that it is recorded that a certain number of insane patients improved while suffering from the acute disease, and that some remained well. I have voluminous tables of cases, but have only submitted to you examples of the forms of disorder which I have met with. I cannot sum up my experience better than by quoting that of Dr. Leledy, Interne at the asylum of Bourges.

Influenza, like other fevers, may set up psychopathy,
Insanity may come on at various periods of the disease.

It may start any form of insanity.

No specific symptoms result from influenza.

The rôle of the influenza varies in the production of insanity.

It may be the predisposing or the exciting cause.

In all cases there is some acquired or inherited predisposition.

The insanity follows from altered brain nutrition, possibly toxic.

Onset of insanity, often sudden and bearing no relationship to the severity of the influenza.

The curability depends on the general rather than special conditions.

The insane are less disposed to take it than the sane.

Rarely it has cured psychoses.

The insane may have mental remission during the influenza.

There is no special indication in the treatment.

Influenza may lead to crimes and medico-legal issues.

The PRESIDENT, in opening the discussion, said that the authors had indicated for discussion the following principal points :—On Dr. Althaus' paper : 1. Influenza was an infectious nervous fever caused by a special poison (grippo-toxine) circulating in the blood, and causing congestion of the medulla oblongata. 2. Perfect or imperfect recovery from the attack of influenza was owing to a sufficient or insufficient quantity of an antidote (anti-grippo-toxine) being formed in the serum of the patient. 3. Immunity once acquired might be lost again by the disappearance of anti-grippo-toxine from the serum. 4. Grippo-toxine resembled the syphilitic virus in its tendency to attack all parts of the nervous system after the attack was over, but surpassed the syphilitic toxine in virulence and rapidity of action. 5. The three varieties of influenza—nervous, catarrhal, and gastric—were not distinguished from one another by any pathological characters, but only by the localisation of grippo-toxine in different areas of the bulb. 6. The nervous form of grip was owing to

congestion of the thermolytic, cardiac, and other centres in the bulb. 7. The catarrhal form of grip was owing to congestion of the nervous mechanisms formed by the nuclei of the fifth pair and the vago-accessory nerves in the bulb. 8. The gastric form of grip was owing to congestion of the vomiting centre in the bulb, the shock being sometimes transmitted to the splanchnic nerves, which anastomosed with the pneumogastric in the cœliac plexus. 9. There were afebrile cases of influenza, the principal symptom being intense mental depression, leading sometimes to suicide. 10. Until the anti-grippo-toxine should have been isolated, revaccination with animal lymph appeared to be the best preventive of influenza. On Dr. Savage's paper: 1. Certain nervous symptoms, such as delirium, insomnia, and neuralgia, were common in influenza. 2. Similar symptoms in an exaggerated form might follow influenza at an indefinite period after the disorder. 3. True insanity rarely followed influenza, unless the patient were strongly predisposed to neuroses. 4. Melancholia was the most common form of alienation, though every other form had been met with. 5. In certain conditions influenza set up general paralysis of the insane. Epileptiform attacks might follow influenza. 6. Influenzal neuroses were fairly curable.

Dr. SYMES THOMPSON considered that Dr. Savage had proved the *post-influenzal* neuroses that fall under the notice of alienial physicians are not accidental sequences, or merely post-febrile affections, but that they are distinctly *propter-influenzal*. That a neural person should set up epilepsy and general paralysis is remarkable, but that it should occasionally cause temporary relief of insanity was in accordance with the recognised fact that shock of any kind was often remedial in insane states. From the 'Annals of Influenza' (written by his late father, Dr. Theophilus Thompson, F.R.S.), it appears that Dr. Graves, writing of the epidemic of 1836-7, attributed the dyspnœa of influenza, which was often out of all proportion to the physical signs, as due to a depression in the vital activity of the lung, and referred to German experiments on the fifth pair of nerves as causing paralysis of lung with marked dyspnœa. Graves considered that the poison acted on the nerves in general and specially on those of the lung. Dr. Peacock also, in his report on the epidemic of 1847-8, concludes that the phenomena of influenza are due to a deleterious influence acting on the nervous system, damaging its vitality, and so poisoning the blood as to predispose to local congestions and complications. Dr. Symes Thompson referred to cases under his own notice in which death had occurred from nerve shock, before there had been time for the development of lung inflammation. These cases had arisen from premature exposure, and tended to support Dr. Althaus' views concerning anti-grippo-toxine. The cases of sudden death from cardiac failure and of exceedingly rapid action of the heart pointed to defective enervation, and the instances in which gastric complications occurred were so much like cases of sea-sickness (desire for death as a relief to misery being conspicuous in each) as to suggest that the nucleus of the pneumo-gastric nerve was involved in either case. With reference to Dr. Althaus' suggestion that the influenza microbe was like the pneumo-coccus, and developed a toxic alkaloid or albumose, which, after a time, produced a neutralising and allied alkaloidal body, this was based on the assumption that such a microbe existed. Until this was discovered, isolated, cultivated, and tested, we must regard the suggestion as no more than a "working hypothesis" which, however, should be hailed, as it tended to give definiteness to our pathology and hopefulness to our therapeutics.

Dr. SANSOM said that many of the points which had been raised had a strong practical importance. He desired to refer especially to the resemblance of the influenzal virus to that of syphilis. It had not fallen to his lot to see a large number of cases of acute influenza, but he had seen many of the results of that disease, and at first he was much puzzled by some of them. A man was seized in the night with intense pain in the liver region, which he thought was colic. It lasted for an hour, and was extremely severe. He had previously been healthy. Next day an enema acted well, and there was no jaundice. The following night he had a similar attack, and the gall-bladder could be marked out by the area of tenderness. After this attacks occurred both by day and by night, and he got relief from opium and belladonna. There had been an antecedent history of influenza twenty months before—a severe attack, accompanied by great prostration, but followed by a period of average health. A number of other cases presented this pain, a form of visceralgia. It was a severe epigastric pain, sometimes constant and sometimes intermittent. The time that these symptoms developed after the initial attack of influenza varied a great deal, from a few weeks to some months. About the same period he began to notice cases in which there were heart symptoms of great severity, like angina pectoris. An athletic man with no evidence of too high arterial tension, was suddenly taken with a screw-like pain at the heart which caused him to fall prone in a state of syncope; the fit was not an epileptoid one. In another case, a patient without an anterior history of influenza became from a heart attack faint to absolute unconsciousness, and afterwards had maniacal attacks. These he regarded as intense attacks of cardialgia, showing no sign of true angina, no high arterial tension, no diseased arteries, no signs of coronary failure. In some cases there was less pain, but there was evidence of perturbation of cardiac action, an extremely rapid or an extremely slow heart occurring as a remote effect. Some had a sense of impending death, without any great pain; he had not known such a case to eventuate fatally. In other cases pain in local nerves was extreme, especially in the calves. A child he had seen with complete motor paralysis of both legs, but she made a perfect recovery. These appeared to him to be instances of peripheral polyneuritis and quite like alcoholic neuritis. One patient had double supra-orbital neuralgia, another had violent right-sided infra-maxillary pain, both being subsequent to influenza. His conclusions were that these lesions which he had instanced were not general or due to influence on nerve centres (though in early influenza the lesion might be a central one), but that the evidence pointed to a local peripheral lesion. The hepatalgia, the cardialgia, and the local muscular pareses were to be explained in this way, and some cases were instances of pure local neuritis, like those produced by alcohol or by the toxins evolved under epidemic influences.

Dr. BEZLY THORNE said that, in rising to thank the President and Fellows of the Society for their courtesy in inviting him to join in the interesting discussion in which they were engaged, he could not help reflecting with some gratification that, whereas, when he had the honour of addressing the Fellows in March, 1890, he stood alone in advocating the essentially cerebro-spinal character of influenza, he found on the present occasion no dissentient voice raised against that hypothesis. Coming to the subject immediately engaging the attention of the Society, he said that he could not agree with Dr. Althaus that the initial incidence of influenza was limited to the medulla oblongata. In his view, the entire cerebro-spinal system of nuclei was invaded at the outset. It had been

his custom, ever since the early days of the epidemic in 1889, to practise percussion of the spinal region in persons affected with that malady, and, as he had stated elsewhere, he had found that marked spinal sensitiveness became rapidly developed in about two-thirds of female patients and in about one-third of males. Indeed, he had noticed that, in the case of a powerful and previously healthy young man, the lumbo-sacral region had become acutely sensitive to percussion within a few hours of the access of the malady. He contended that the essential and most important phenomena were not, however, to be looked for in the course of the pyrexial stage but in the period of vital depression which ensues and lasts for a period extending from a few days to weeks or even months, and of which the fundamental pathological condition appeared to be a dilated condition of the minute vessels of the cerebro-spinal centres and consequent functional impairment or perversion of those important organs. Among the more noteworthy features of that state of vital depression, he said, were loss of flesh and body heat, and he considered the condition essential to safety and early convalescence to be rest in the horizontal position until such time as those two leading symptoms had yielded to treatment and the recuperative powers of nature. Much evil ensued from hurrying the supposed convalescent into taking the air in wheeled conveyances, and thus subjecting the nerve centres to numberless disturbing secussions and vibrations. In fact, the subject of influenza required to be treated on the same general principles as were recognised to be indicated in cases of cerebro-spinal concussion. He enforced the necessity of rest by quoting the case of a lady whom he had seen immediately after a short railway journey, and from the appearance of whose eyes he had immediately recognised that she was in the initial stage of influenza. She had been most carefully tended, and had strictly maintained the horizontal position for a week without so much as once placing her feet on the ground. At the end of that interval he had given permission for her to spend a few hours a day in an adjoining room. On seeing her again on the third day he had been startled by her pinched and wasted appearance, and found her temperature, which had previously risen to the normal point, to be no more than 94.8° . She admitted that, since she had left her bed, her clothes had come to hang on her "like a bag." The use of the thermometer was doubtless interesting, and, in a degree, useful during the pyrexial period, but yielded indications of primary importance during that stage which is too often regarded as one of convalescence, but is, in fact, the really grave part of the malady, that in which the poison, having invaded the nerve tissues, remains in dangerous and often persistent occupation. Could it be surprising that patients in such a condition succumbed in a few days or even hours to complications which, under less serious conditions, would present no danger to life? Dr. Thorne further gave it as his opinion that the sequelæ commonly observed in influenza are not solely attributable to central nerve causes, but mainly determined by pre-existent or intercurrent conditions. For example, the man who found himself to be attacked while following his avocation, and, in returning home, was exposed to the inclemency of the weather, would be the most likely to develop peri- or endo-carditis, pleurisy or catarrhal pneumonia; and the subjects of unhealthy tonsils, of eustachian, laryngeal, or gastric catarrh, would be those most likely to suffer from complications in those particular regions which were thus predisposed to become the seat of congestion or inflammation.

Dr. SISLEY asked Dr. Savage if he could explain how it was that "the

insane are less disposed to take it (influenza) than the sane." In making an enquiry as to the mode of spread of influenza, he had been struck by the fact that isolated communities suffered much less from the disease than people who mixed with the world. Thus, in the epidemic of 1889-90, the report of the Commissioner of Prisons showed that the prisoners in seven out of a total of twenty jails entirely escaped infection, although influenza was prevalent in the towns in which the jails were situated. He thought this was due to the fact that prisoners lived retired lives and therefore ran less risk of contagion. He asked Dr. Savage whether the comparative immunity possessed by the insane existed in those who were at large, as well as those in asylums. It occurred to him that the enforced isolation of asylums would lessen the spread of influenza by contagion, and asked Dr. Savage whether he thought the fact he had stated should be explained in that way, or whether there was any peculiar resisting power in the insane diathesis.

In referring to the paper of Dr. Althaus, Dr. Sisley pointed out that, although, doubtless it was based on well-ascertained pathological facts, these were not given; his remarks, therefore, would be chiefly directed to mentioning the points on which more information was wanted. Before doing so, he strongly protested against any new name being given to influenza; the disease had already enough, and more than enough names, and when a new name was given to an old disease, as Dr. Wilks had said, "the only advantage is to the man who names it." He hoped the Society would not countenance any such innovation as that proposed.

Dr. Althaus defined influenza as "an infectious nervous fever caused by a special poison—grippe-toxine—circulating in the blood, and causing congestion of the medulla oblongata." This definition could not be considered a satisfactory one, and he asked Dr. Althaus what evidence he could adduce that the disease *was* caused by congestion of the medulla oblongata.

Dr. Althaus compared the poison of influenza with that of syphilis. There was, however, little similarity in the action of these diseases on the nervous system. Syphilis affected the nervous system very late in the disease, generally years, often many years after the primary lesion. In influenza the nervous system was affected immediately. For example, in one of the first cases he had seen in 1889, the onset of the attack was so sudden and the loss of power so complete, that the first doctor who saw the patient, diagnosed the case as one of myelitis. Cases were on record of strong men—miners—suddenly prostrated with the disease and unable to walk. He had heard of no case in which syphilis had so quickly acted on the nervous system. In syphilis, again, the organic pathological changes in the nervous system and in the viscera were well marked and distinctive. Were there distinctive pathological changes in the nervous system in influenza? If so, what are they? It seemed to him that the latter nervous manifestations of influenza were more easily comparable with those seen in diphtheria; and in both these diseases the symptoms were usually of a transitory character, which was unfortunately often not the case in syphilis.

Dr. Althaus took exception to the generally received classification of cases of influenza into nervous, catarrhal, and gastric. This was not a pathological, but a clinical classification, and as such had been found convenient by the physicians of all countries. But it was not true to say that the catarrhal form of influenza produced no pathological changes. The changes were peculiar, if not distinctive. They were described years

ago by Gluge, and Grasset had recently given an account of them, which corresponded exactly with the cases he had seen here. The appearances were like those seen in the broncho-pneumonia of children; there were scattered patches of pneumonic consolidation.

Dr. Althaus had said that "the nervous form of grip (influenza) is owing to congestion of the thermolytic cardiac and other centres in the bulb." What anatomical evidence did he bring forward to support this?

The catarrhal form, Dr. Althaus said, is due to "congestion of the nervous mechanisms formed by the nuclei of the fifth pair and the vago-accessory nerves in the bulb." What anatomical evidence did he adduce to support this statement?

"The gastric form," Dr. Althaus said, "is owing to congestion of the vomiting centre in the bulb." What evidence was there of this statement? Had Dr. Althaus seen such a case or had one been recorded? Were these statements, in fact, the result of observation, or were they pathological speculations?

For the prevention of influenza many things had been recommended; the best way in which the disease could be avoided was to avoid the risk of infection. Dr. Althaus recommended vaccination with animal lymph. The suggestion was an interesting one. But on what grounds was it made? It was said that German soldiers were re-vaccinated, and that German soldiers suffered less from influenza than the civil population. The statistics of the incidence of influenza amongst the soldiers were, doubtless, correct. But what method had been adopted for estimating the amount of sickness amongst the civil population? Even if a correct estimate had been arrived at, it was useless to consider it without taking into account the difference in the conditions of life existing between the classes compared, and for statistical purposes it would be necessary to compare the incidence of the disease in recently re-vaccinated soldiers with healthy unvaccinated males, living under similar conditions. It was generally believed that one specific disease did not give immunity from another, and that if it were proved that vaccination protected from small-pox and from influenza, current ideas on the whole subject would have to be modified.

Dr. Althaus had given names to certain substances which he said caused and gave immunity from influenza, but as these substances had not been found, it seemed premature to give them names; should any one find them, the discoverer might wish to name them himself. These theoretical substances, Dr. Althaus supposed, were due to the work of an undiscovered microbe, and as a first step it seemed necessary to find the microbe. Dr. Althaus' suggestion reminded him of a saying attributed to the late Mrs. Glass, with respect to the cooking of a hare, "First catch your hare," she is reported to have said. Dr. Althaus kindly suggested that some junior Fellow of the Society should find the microbe, but there appeared to be no reason why such a research should be confined to the junior Fellows. In conclusion, he begged Dr. Althaus to give fully the pathological observations on which he had based his statements that influenza was due to congestion of various nervous centres, and to say whether he had found them in the course of his own researches, or where they were recorded.

Dr. HERON said he had seen a good many cases of influenza, and amongst them there were very few complications. In fact, had it not been for two cases which came under his notice, he would have had to say that he had seen no complication which could have been rightly described as serious in any degree. As Dr. Savage had pointed out, there were many medical

men who had seen a large number of cases of influenza, without having had, among these cases, any experience whatever of grave complications associated with or following upon the attack of this disease. The two cases of severe complication following attacks of influenza which Dr. Heron had seen, were practically identical in kind, though the one which he described was much more severe in its chief symptoms than the other one had been. The case was that of a man, between 40 and 50 years of age, who suffered from an attack of influenza of seemingly the ordinary type. As the feverish features of the disease passed away, the patient became markedly depressed in mind, and complained of an overpowering sense of physical languor. The mental symptoms deepened, and this man became really melancholic. The patient was himself the first to notice that he was actually suicidal; and he took pains to avoid having at hand the means of self-destruction. This condition sometimes deepening, sometimes almost passing away, persisted for nearly four months, and then gradually cleared up. The important practical point about this case was that it illustrated the line of practice which should be followed, in Dr. Heron's opinion in all cases of melancholia. These cases should, he urged, always be regarded, from the point of view of treatment, and in the sufferers' interests, as prone to suicidal impulse. It was not the case that every instance of even true melancholia was of suicidal tendency; but the risk was always so tremendous that he thought it should be a rule of practice to regard all melancholic persons as of suicidal tendency. Dr. Heron asked for Dr. Savage's opinion on this point. Another matter of, possibly, great practical importance in connection with influenza, was the fact, that a considerable number of instances seemed to show that the poison of this disease was apt to infect persons living in certain rooms or part of a house, while the people living in other parts of the same house escaped the disease sometimes altogether. This had been illustrated in various hospitals and asylums; amongst others, at the Glamorgan County Asylum, where the male patients and attendants alone suffered from influenza; and at one of the large hospitals in Berlin, where the patients who suffered from the disease were all inmates of certain wards in the hospital. Also the disease seemed to have a tendency to recur in rooms, or parts of a large building, where it had previously been. This last fact was being illustrated in the course of the present epidemic of influenza; the disease having reappeared in certain rooms, and wards of hospitals where it had first appeared during the epidemic of last year. These facts seemed to indicate the importance of seizing at once upon the persons and places first infected with influenza, and carefully treating them by means of disinfection and isolation, in the hope of stopping the spread of the disease amongst the healthy population.

Dr. LEONARD GUTHRIE was pleased to hear Dr. Sisley compare the neuroses of influenza with those of diphtheria. The resemblance was not merely a coincidence, it depended upon the similar action of special poisons in the same areas or cells, in the central nervous system. The action of each poison seemed to be twofold:—(a.) *Irritant*. (b.) *Depressant*. In the case of influenza, the *irritant* action was in excess, whilst in the case of the diphtheritic paralysis the *depressant* action was predominant. The following instances were given of the analogous nervous symptoms which attend each disease:—(1.) *The Pulse*.—One of the earliest symptoms noticed in influenza was bradycardia, which existed not only during the height of the fever but remained for days, weeks, or months after the crisis. Mr. Marks (?) had mentioned fatal cases of this sort. In diph-

theritic paralysis, bradycardia was also not uncommon. Cases were mentioned in which the pulse, after diphtheria, became slower and slower until it finally stopped. On the other hand, in both diseases, bradycardia sometimes gave way to the opposite condition, "tachycardia," in which the pulse became extremely rapid, weak, and sometimes irregular and intermittent under these circumstances. The slightest movement of the head or body, or even a mental emotion, was sufficient to bring about a fatal cardiac crisis. It was suggested that bradycardia in these cases was due to irritation of the cardiac inhibitory centre of the vagus, whilst tachycardia depended upon depression or paralysis of the same centre. (2.) *Pulmonary Complications*.—True lobar pneumonia was uncommon in both diseases, but when it occurred, it was so extremely fatal, as compared with ordinary lobar pneumonia in a subject previously strong and healthy, that some other cause of death must be sought in these cases. Probably the lobar pneumonia was secondary to paralysis of the pulmonary vagus.

Broncho-pneumonia was very common. In the case of diphtheritic paralysis, it was believed to be secondary to pulmonary collapse, and the collapse was due to paralysis of various portions of the chest wall, especially of the diaphragm and, perhaps, also of parts of the pulmonary vagus. Dr. William Pasteur believed that pulmonary collapse followed such paralysees, and he supported his views by reference to the pulmonary collapse which Drs. Martin and Hare, of Philadelphia, had shown to follow section of the phrenic nerves. The fleeting patches of consolidation in the lungs, described by Dr. Althaus in cases of influenza, might also be due to temporary collapse of the pulmonary tissues, and consequent broncho-pneumonia. *Bronchorrhœa* and *hypersecretion* of mucous membranes. Dr. Althaus had described the profuseness and suddenness with which this took place in influenza. In diphtheritic paralysis the same condition occurred. Mucus and saliva poured from nose and mouth, the eyes became glazed with conjunctival secretion; ulcers formed in the tongue and lips, and the lungs, in the space of a few hours, became water-logged with bronchial and oral fluids, so that the patient sometimes died literally drowned in his own secretions. Death from capillary bronchitis was usually certified in these cases, but the condition was, doubtless, paralytic. The term "bulbar cases" had been given to symptoms pointing to sudden disturbance or paralysis of all or most of the bulbar centres in diphtheritic paralysis. Akin to these were the cardiac, respiratory, and gastric crises of influenza. Gastric crises in both diseases were associated with severe epigastric pain, incessant vomiting, and alteration of pulse rate. Dr. Gee had collected several cases of incessant vomiting after diphtheria, and he was inclined to attribute the symptoms either to uræmia or to neuritis of the vagus. But albuminuria was frequently absent, and Dr. Guthrie preferred to regard them as due to the irritation of the vomiting centre in the bulb rather than as evidence of neuritis. *Post-mortem* congestion was of little value as evidence of causation of symptoms during life. *Eye symptoms*.—Various oculomotor pareses, sometimes amounting to complete ophthalmopagia with loss of accommodation, have been described by Dr. Greef as results of influenza. These were the common eye conditions which followed diphtheria. Probably in these cases there was paralysis of the eye centres beneath the corpora quadrigemina. The functional nature of their neuroses formed another link of resemblance between influenza and diphtheritic paralysis. A common sequela of influenza was neurasthenia, with its general, mental, and bodily depression—vague aches and pains, sensory disturbances, and wandering pareses.

Dr. Stretch-Dowse had compared the neuroses of diphtheria with those of hysteria, and Dr. Gowers had said of diphtheritic paralysis :—"Irregular waves of palsy seem to flow throughout the body, sometimes quickly, sometimes slowly, their influences we cannot discern, their course cannot be foretold." This description surely implied modification of function rather than gross organic changes in the nerve centres affected. As a minor point of resemblance might be mentioned, the condition of the knee-jerk in these diseases. Exaggeration was common in hysteria and post-influenzal neurasthenia, but sometimes the knee-jerk was absent. The common loss of knee-jerk in diphtheritic paralysis was almost invariably preceded by excess. One explanation, but not the only one was, that irritation of the anterior corneal cells in the spinal cord, caused the excess of knee-jerk, whilst depression of their activity accounted for its absence. Want of time prevented more than a brief allusion to the comparison which might be drawn between the attacks of aphonia, dyspnoea, orthopnoea, inability to swallow, cardiac palpitation, and incessant vomiting, of hysteria, and the similar attacks which occurred in influenza and diphtheritic paralysis. As examples of extreme irritation of bulbar centres by poisons, might be mentioned, the results of tetanus, hydrophobia and strychnia. He concluded that if Dr. Althaus' explanation of the various manifestations in influenza be accepted, a similar, though modified, explanation, accounted for the analogous symptoms which occurred in diphtheritic paralysis and perhaps in other nervous diseases dependent upon toxæmia.

Dr. RICHARD PARAMORE wished to draw attention to what he considered a great danger to which patients suffering from influenza are liable, although apparently doing well, viz., that of hyperpyrexia, which fact had not been alluded to, or at any rate emphasized by former speakers. He instanced a case of a young woman, aged twenty years, who had always enjoyed good health, but in whose house there had been previously cases of influenza which had recovered. Whilst at her place of business she felt ill, and on her way home called upon him. She was told she had influenza, and was advised to go to bed. On the following morning she was seen, and her temperature was 104° ; she also complained of insomnia, for which chloral and bromide of potassium were ordered. On the third day she appeared better, and had a temperature of 102° . On the fourth day her temperature was 101° , and she was cheerful, and was sitting up in bed. On the fifth day her temperature was normal, and no hypnotic was deemed necessary, but quinine was ordered. However she had a restless night, and the sixth day, when he saw her, she was moribund, with a temperature of 111° . Cold water cloths and ice were applied externally, which reduced the temperature to 108° , but she died about an hour after his arrival. As there was no lung or other apparent complication, and as the patient, who was considered the strongest of her family, was able to take plenty of nourishment, he did not anticipate such an untoward result.

Dr. ALTHAUS, in reply, said that, as no other theory had been brought forward in opposition to his own, he concluded that it had proved acceptable to the Fellows of the Society; and he claimed for it that it gave a physiological explanation of all the multifarious symptoms, complications, and sequels, which were liable to appear in connection with grip. In reply to Dr. Sansom he stated that, although peripheral nerve-lesions were common after influenza, affections of the nervous centres were likewise apt to occur in connection with it, even in persons in whom no neurotic

history or predisposition could be ascertained. He mentioned cases of general paralysis of the insane, spastic spinal paralysis, and locomotor ataxy which he had seen, as direct sequels of the distemper, and contrasted the rapid evolution of these diseases, when owing to influenza, with their much more protracted course when following syphilis. In reply to Dr. Bezly Thorne, who maintained that the disease was not central in the beginning, he pointed to the extraordinary degree of prostration which was one of the most prominent symptoms of grip, and which had arrested the attention of the older observers, who had, however, simply registered the fact that the exhaustion of the patient suffering from grip was out of proportion to the strength and duration of the fever, but had not attempted any explanation of it. Dr. Althaus thought it was easily accounted for by his theory that the poison of grip attacked with preference the very sources of life—Ploureur's "*nœud vital*"—viz., the cardiac and respiratory centres in the bulb; and this localisation of the poison made it intelligible why the whole system of a man might be thoroughly demoralised by such an attack. He contrasted the sudden prostration felt on the first day of grip with the state of things in the first week of typhoid fever, where the patient often simply felt out of sorts, and was astonished when the doctor ordered him to bed. In influenza the patient sought the bed at once, without waiting for the doctor's instructions. Replying to Dr. Sisley, he stated that autopsies showing congestion of the bulb in grip were still wanting, but trusted that this condition would in future be looked for and discovered.* It was not correct to say that nervous affections after influenza were transitory in their character, as many persons had died of hæmorrhage, softening, inflammation, and abscess of the brain, and others had become apparently permanently insane.

Dr. SAVAGE thanked Dr. Symes Thompson for his kindly and appreciative remarks on his paper. He fully recognised that not only with influenza but with other febrile diseases there may be temporary amelioration in the insanity during the fever, and only that day he had seen a lady who, being subject to neurasthenia before she had influenza, was for a time much better of her nervous symptoms, while suffering in bodily health. Dr. Thompson's experience agreed with his as to the frequency with which hereditary predisposition occurred in cases in which neuroses followed influenza. The historical references of Dr. Thompson all confirmed his impression that in previous epidemics there was generally noticed similar nervous exhaustion. Sir Henry Holland recognised not only this, but suggested an atmospheric cause of the disease. He (Dr. Savage) had on several occasions been struck with the rapidity of the pulse in cases of post-influenzal melancholia; in fact, not having heard of an attack of influenza, he had in some such cases, at once said, "Has not the patient suffered recently from influenza?" with the constant result that this had been found to be the case. Dr. Sisley wanted to know whether he thought insanity a kind of prophylactic against influenza. He did not, but he thought the fact that the insane in asylums suffered less than did the attendants was an argument in favour of the propagation of the disease by contagion. In one large asylum in the north, 50 per cent. of the attendants suffered, whereas only 10 per cent. of the patients were

* Intense congestion of the bulb, almost amounting to capillary hæmorrhage, has since then been discovered in a fatal case of influenza by Dr. MacDonald, of the Dorset County Asylum ('*Lancet*,' March 12, 1892).

affected. Dr. Heron had not himself seen many cases of insanity following influenza, but this was due to the fact that whereas he saw the severe cases, in the earlier stages, he (Dr. Savage) saw such cases long after, when they had passed from under Dr. Heron's care. As to suicide, he feared that every melancholic case, and of such the number was very great, following influenza, should be looked upon as possibly suicidal, and treated as such. Almost daily he had had evidence of suicides resulting from post influenzal depression.

November 9th, 1891.

REMARKS ON THE CONDITIONS OF CURE IN CONSUMPTION.

By I. BURNEY YEO, M.D., F.R.C.P.

ABOUT a year ago the medical profession throughout the whole world were excited beyond measure by the authoritative publication of a statement that a cure for consumption had been discovered. Since then the question of the discovery of a cure for consumption has been the subject of frequent debate in nearly every city in the civilised world. This, then, seems an opportune moment to inquire, What are the conditions of cure in consumption?

The curability of consumption is universally admitted, yet, on the other hand, there is a widespread belief, founded on an overwhelming weight of evidence, that consumption is a very fatal disease, rarely cured, almost incurable. Morbid anatomists report numerous instances of the discovery, in the lungs of persons dying of other diseases, anatomical evidence of arrested and cured pulmonary tuberculosis, whereas it is undoubtedly difficult, even for those who have had the largest clinical experience in the observation of cases of consumption, to point definitely to many cases of the undoubted permanent cure of undoubted phthisis.

These are propositions which will command general assent, which are certainly true, although somewhat opposed to one another. We all admit the curability of pulmonary consumption,

but we are also compelled to admit that phthisis, as we encounter it in its clinical forms, is rarely cured.

It is almost impossible to trace out the past clinical histories of those persons who, on *post-mortem* examination, are found to have had, at some period of their lives, pulmonary tubercle which has been arrested and cured. Many probably had no clinical manifestations of phthisis; their tuberculous affection gave rise to no notable symptoms, was wholly latent, and disappeared, leaving only an anatomical record of its existence. They were never consumptives or phthisical in the clinical sense of the word, and it would be more correct to call them cases of cured or arrested pulmonary tuberculosis than cases of cured phthisis. Quite recently—since, indeed, this paper was written—Dr. Coates, Dr. Fowler, and Dr. Sidney Martin have described the anatomical appearances presented by some of those so-called obsolete or cured tubercles, and they are thus described by Hérard and Cornil.*

“A few pigmented granulations, which are the vestige of a very localised and unimportant tuberculosis, which is extinct, if not altogether cured. The fibrous tissue, and the giant cells which it encloses, appear to be tolerated without of themselves giving rise to any injury. That is a form of tuberculosis which may pass as curable or cured. It is sometimes met with in the necropsies of individuals who have died accidentally of some quite different cause. . . . These fibrous or calcified tubercles contain very few or no bacilli; bacilli have, however, been occasionally found in calcified tubercle. . . . Nothing is more common than to find at the apices of the lungs of old men, which are almost always indurated, these hard nodules or cysts filled with whitish or yellowish chalky substance; but it would be an error to refer all those changes indiscriminately to cured cavities, for they may quite as well be simply bronchi, somewhat dilated, which have become isolated by an interstitial pneumonia, or cured abscesses resulting from a simple pneumonia or gangrenous foci, or productions of a syphilitic nature. All lesions which have nothing characteristic in themselves in the stage of fatty or cretaceous change in which they are found;” but they admit that “a great number of these cases may in reality be cured phthisis.”

Professor Brouardel, the eminent medical legist states, that he has found evidence of cured tubercle, cretaceous, fibrous, or

* ‘La Phtisie Pulmonaire,’ 2nd edition, p. 227.

cicatricial, in the apex of the lungs of 60 per cent. of those over 30 years of age whom he has had to examine *post mortem* on account of their having died a violent death. "Yet," adds Professor Grancher, who quotes these statistics, "we are powerless to cure the tuberculous who have become phthisical," the exceptional instances only proving the rule. Thus we have put before us two propositions, in appearance irreconcilable. According to the first, pulmonary tubercle is often cured; according to the second, it is incurable. The explanation lies in the fact that these cases of spontaneous cure of tubercle have never been cases of phthisis. "In almost all these cases the tubercles are small and separate, incapable, in the state in which they have been found, as well as in the course of their development, of modifying the respiration sounds or vibrations. They are generally small masses of the size of a pea, at the extreme apex of the lung, and surrounded by healthy, crepitant lung tissue, or they are isolated, fibrous tubercles scattered in small number in the centre of the lung."*

Morbid anatomists appear by no means agreed amongst themselves as to the frequency with which these evidences of cured pulmonary tubercle occur in the lungs of persons dying of other causes than phthisis. I have just quoted Brouardel's estimate; Dr. Coats estimates it at 25 per cent., Dr. Fowler at 9 per cent., Dr. Sidney Martin at $9\frac{1}{2}$, and the Vienna statistics quoted by Dr. Fowler at 4 per cent. only. Now, with such very wide differences in the estimates of different morbid anatomists, it would seem probable that some accept as instances of cured tubercle cases that would not be accepted by others. It must, however, I think, be admitted (1) that pulmonary tuberculosis in certain forms and under certain conditions is commonly and spontaneously cured; (2) that phthisis is rarely cured, meaning by phthisis pulmonary tuberculosis that has reached such a degree of development as to seriously affect the general health, and to give rise to easily recognised physical signs. Still, cases of the latter kind are occasionally, if exceptionally, cured. It is, then, a problem of great practical importance to ascertain what are the conditions which determine the cure of pulmonary tuberculosis in the pre-phthisical stage, as well as those which lead to the less frequent but occasional arrest and cure of the phthisical state itself. Now, it is an obvious fact, yet one too commonly lost sight of, that the

* Grancher, 'Maladies de l'Appareil Respiratoire,' p. 140.

cure of all disease is conditioned. So, in applying tests to proposed remedies, we should not be forgetful of the fact that no remedy can cure a disease unless the conditions of its cure exist.

Not long ago I took occasion to remark in one of the medical societies of London, only a few weeks before Koch's announcement of his discovery of a cure for consumption, that if a perfectly certain cure for tuberculosis were at any moment to be discovered, it would leave the majority of the then existing cases of consumption incurable. Why? Because the conditions of cure no longer exist.

Yet whenever a new method of treatment or a new remedy for consumption is announced, it is tested—seriously tested—not only in cases in which cure is possible and conceivable, but in a far greater number of cases in which cure is impossible and inconceivable; in which the conditions of cure have long ceased to exist. I say “seriously tested” because those hopeless cases are brought into the statistics published, and are used to discredit the remedy or mode of treatment. It is then, I repeat, an important practical consideration to ascertain what are the conditions which favour the cure or arrest of tuberculous disease of the lungs, or of consumption.

In the first place, every one is agreed that early recognition of the disease is one of the chief conditions which render a cure possible. If we are able to treat the patient before his pulmonary tuberculosis has developed into phthisis, we may hope to contribute to bringing about the same arrest and cure as Brouardel and other morbid anatomists assert to be so common.

The evolution of phthisis is, in most of the common chronic forms, exceedingly slow; and months, and even years, may pass before the isolated tuberculous lesions mass together or “conglomerate,” and become easily detectable by physical signs.

Laennec said that a patient does not die of his first attack of tuberculosis, and Bayle recognised a pre-phthisical period which he termed “occult phthisis,” or “the germ of phthisis.” “In many instances,” he says,* “before the manifestations of the first symptoms, there is an interval during which the patient whose lung is already profoundly injured, appears still to enjoy the best of health.” The purely arbitrary and somewhat unnatural division of pulmonary tuberculosis into first, second, and third

* Bayle, ‘Phthisie Pulmonaire,’ Paris, 1810.

stages, with their respective physical signs, has been to some extent responsible for the prevalent neglect to recognise this earlier stage of the disease.

As the recognition of this early period of pulmonary tuberculosis is one of the chief conditions of its cure, I may, perhaps, be permitted to dwell briefly on this "occult stage," or "stage of germination," of consumption, as it has been called.

"From the first appearance in the pulmonary parenchyma of the first embryonic cells (the result of bacillary influence) up to the production of the adult miliary tubercle, there is a whole series of stages which together constitute a period of formation or germination. If during this period a section be made of the apex of the lung we encounter some tubercles in the follicular stage (microscopic) and a few others still less advanced, scattered through certain lobules, the surrounding lung being healthy or slightly congested."*

This is the anatomical condition. Is there a recognisable clinical condition corresponding to it?

"In an individual," says Professor Grancher, "suspected or not of tuberculosis, a few tubercles begin to develop slowly; embryonic cells and giant cells form and group themselves together, so as to constitute the follicular tubercle—that process lasts a long time and is silent—and nothing appears changed in the health of the man doomed to phthisis; sometimes a little anæmia, a little debility, a slight cough, a little quickened respiration—nothing more."

Yet it is to the diagnosis of this period that all our efforts should be directed. Is such a diagnosis possible? Yes: to the extent of probability, not to the extent of certainty. Certainty is only reached when the bacillus can be detected in the expectoration, and the bacillus cannot be detected in the expectoration until there is destructive ulceration of the lung and a small cavity formed communicating with a bronchial tube; in short, not until the phthisical process is established and somewhat advanced. We have to be content, then, with a probable diagnosis, and we may now consider how such a probable diagnosis can be established. We must notice closely and carefully any slight modification or abnormality in the respiratory sounds, particularly when such abnormality, however slight, is localised and fixed. Harsh, low-

* Grancher, *op. cit.*, p. 136.

pitched respiration, and especially inspiration, is the most important; feeble, incomplete respiration when combined with inspiratory harshness, and jerky or interrupted inspiration are often also important indications of early tuberculous changes in the apex of the lung. It is to the fixity and localisation of these slight anomalies of respiration that weight must be attached. When such modifications of the respiratory sounds are accompanied by some evidences of disturbance of the general health, the diagnosis of an early stage of pulmonary tuberculosis becomes extremely probable.

The early occurrence of hæmoptysis is a condition I am disposed to consider favourable to the cure of phthisis. It is so, I believe, because it calls attention, in a striking and impressive manner, to this early stage of the tuberculous affection, when otherwise it might pass unobserved and undiagnosed.

On the other hand, there is, I believe, another condition of the lung, attended with well-marked physical signs, often erroneously diagnosed as the first stage of phthisis, and which goes to swell the number of cases of cured phthisis. I refer to cases of apical, dry pleurisy of rheumatic origin. In such cases you will often meet with a troublesome, dry cough, difficult of cure, and on physical examination you will usually encounter at one or other apex some loss of resonance on percussion, diminished respiratory movement, not infrequently a little retraction of the subclavicular region, a creaking friction sound with the inspiration in the supra-spinous fossa, which disappears after a few very deep inspirations, and either enfeebled breath-sound or occasionally a faint sub-crepitant *râle*. Vocal fremitus is usually diminished, and sometimes absent.

Why, you will ask, should not such cases be regarded as cases of early pulmonary tuberculosis with secondary involvement of the pleura? The reasons are these: In the first place, these cases occur in persons of the rheumatic diathesis, and are often accompanied by other rheumatic articular manifestations; they are usually traceable very distinctly to exposure to chill; there is constantly a history of pain in the region affected at the onset of the attack; and moreover the physical signs would indicate a much more advanced and serious condition of tuberculosis, were they due to that disease, than we have any other evidence of; for there is an absence of fever, of wasting, and of the general features

of phthisis; and finally, these cases are cured by suitable treatment, such as we should apply to rheumatic affections of other parts, although the physical signs of adherent or thickened pleura in many instances remain.

It is true that such cases as I have described require to be carefully protected from the possibility of tuberculous infection, for if neglected or placed under unfavourable conditions the physical changes about the apex of the lung render it vulnerable to bacillary infection. The comparative immobility of the subjacent lung and the consequent inactivity of the respiratory currents and the tendency to congestive and catarrhal conditions which the proneness to blood and air stasis must provoke naturally lead to a susceptibility to the acquisition of tuberculosis.

Another condition which favours the cure or arrest of consumption in the more advanced stage—that is, in the stage of “conglomeration” of tubercles—is a natural tendency in the evolution of the tubercle to fibrous change. Professor Grancher* vigorously protests against Virchow’s definition of tubercle as a neoplasm incapable of organisation. He contends that it has, on the contrary, a natural tendency to fibrous organisation.

“Always formed,” he says, “of two concentric zones, a central caseous and a peripheral of embryonic cells, the tubercle, whatever may be its anatomical form, advances either to sclerosis or caseation, according as the cellular zone or the caseous zone develops with greatest rapidity. That is why so many tubercles are cured spontaneously and by the vigour of the organism alone, which would be impossible if the tubercle did not contain in itself the germ of its cure. The embryonic zone, always present, can repair the destruction and fill up small cavities by cicatricial vegetations; but if from the beginning the tendency to fibrous evolution is greater than that of caseation, a cure may take place without loss of substance by connective vegetation even to the centre of the tubercle, which becomes fibrous or encysted. But if all tubercle may be cured by sclerosis, certain anatomical forms are much more prone to it than others. Compare the ‘pneumonic tubercle,’ so rapidly and easily caseified, with the tubercles of the slow phthisis of old age, fibrous from the commencement.”

Whatever, then, favours the sclerotic or fibrous evolution of tubercle promotes the natural condition of cure.

* *Op. cit.*, p. 244.

The next condition of cure I would mention is probably closely related to the preceding one. I refer to the absence of tissue sensitiveness or irritability—the absence of that tendency to acute inflammatory reaction to the bacillary infection, the presence of which is so unfavourable a prognostic and usually leads to a rapid extension of the local infection and to a diffuse ulcerative destruction of lung tissue. It must have occurred to every experienced physician to see cases of phthisis in quite the early stage, in which the tissue sensitiveness and constitutional reaction were so marked that an unfavourable prognosis is at once forced on the mind. “The organism” (says Grancher) “far from reacting in a uniform manner to the presence of the tubercle bacillus, may in one case yield and break down rapidly, and in another struggle and triumph.” It has seemed to me that individuals who show vascular irritability, who flush readily, and in whom the vasomotor control would seem to be weak, or possibly the vascular walls thin, offer a comparatively feeble resistance to tuberculous infection. The pneumonic form of pulmonary tuberculosis, although one of the most serious, and often rapidly fatal, is not, however, necessarily incurable. There is a benign form, as pointed out by Grancher, in which the pneumonic process is neither very violent nor very extensive, and if the organism retains some vigour and if suitable and early treatment comes to its aid, the softening may be circumscribed and the tuberculisation localised and subsequently cured.

Another condition favourable to cure is the absence of marked hereditary predisposition, the possession of a sound, vigorous constitution which has become accidentally infected by the tubercle bacillus. Another possible condition is a mitigated virulence of the bacillary infecting agent, and the small quantity or number that originally gain access to the lungs. The channels, also, through which the bacilli gain access to the lungs influences the conditions of cure; if they reach the lung through the blood vessels or the lymphatics the conditions will be unfavourable, owing to the probability of a wide diffusion of the infective agent; their entrance with the respired air is a more favourable condition.

So in studying the effect and influence of parasitic agents in producing pulmonary lesions, we must take into account not only the irritability of the tissues attacked, but also the energy of the

irritant. It is by no means certain that all tubercle bacilli are of equal energy, and it is quite possible that the condition of cure in one individual as distinguished from another may be that he has been infected by a parasite much less energetic, much less virulent than the other. This would certainly seem to be the case in some other infective maladies; in the case of the scarlet fever microbe, the remarkable variations in its virulence or energy has long been a recognised fact. But in the case of the tubercle bacillus, it would appear to be more probable that it is in the relative irritability of the tissues of the various individuals attacked that the greatest differences are to be observed. This consideration points to a probable mode in which the efficacy of therapeutic agents may be exerted, namely, by lessening the irritability of the tissues, and so diminishing the rate of progress of the disease, thereby gaining the time necessary for the natural arrest of the morbid process through the evolution of sclerotic changes.

There is yet another most essential condition of cure, and without it most of our therapeutic efforts will end in failure, and that is a sound organic state, and a sustained functional activity of the organs of digestion and assimilation. The phthisical patient who cannot digest and assimilate nourishment is in the greatest peril.

I will in the next place refer briefly to a few of the therapeutic conditions which I believe favour the cure of phthisis. First in order I would place the administration of suitable food, in suitable quantity, and given in such forms and in such methods as shall ensure its digestion and assimilation. Scarcely sufficient attention in detail is given in this country to this very important condition of cure. It is otherwise in France; there the methodic alimentation of phthisical patients enters as a principal factor in their therapeutic management. There the principle of excessive feeding (*sur-alimentation*) in the treatment of phthisis has been pushed to an extent which it would probably be difficult to make acceptable to English patients; for I fear very few of our patients of the better class would consent to have large quantities of concentrated food pumped into their stomachs through a tube. Yet it is insisted upon by very competent authorities in France that, in order to maintain the prolonged and continued labour of cicatrization on which the arrest and cure of tubercle depends, the necessary cellular activity can only be obtained by hyper-nutrition, which can only be procured by hyper-alimentation. "Without,"

says Professor Grancher,* “an altogether special alimentation you are powerless; in short, alimentation is the first factor in the promotion of curative sclerosis.”

The success attending the application of the Weir-Mitchell system of feeding has shown how great an excess of food the most unwilling patients may, by systematic management, be induced to consume and assimilate; and a somewhat similar method has been applied to the treatment of pulmonary tuberculosis, with great success, by some of the most eminent physicians in France. Grancher reports the case of a lady with somewhat advanced phthisis who had so great a distaste for food that for several days she had refused to take any, yet by the diligent and persevering application of a rigorous method they finally succeeded in getting her to take large quantities of concentrated food and stimulants; so that she arrived at the point of consuming at least twice as much food as her sister and husband together; and, without reckoning six tea-spoonfuls of cod-liver oil, which she took in a glass of beer, she absorbed a relatively large quantity of champagne, malaga, and cognac. This patient increased in weight in less than six months from 70 to 105 lbs. She recovered from her phthisis, and died a few years after of cancer of the uterus.

Another condition of cure is the possibility of living a life much in the open air of the country or of the sea; and, if possible, in a climate where there is much sunshine, and where the air is relatively dry and unirritating. A dry aseptic atmosphere especially favours the arrest of pulmonary tuberculosis, and promotes curative changes in the tuberculous deposits. A dry, pure atmosphere—not necessarily a cold one, unless for exceptional organisations. In Western Europe it is difficult to find a dry, pure, sunny atmosphere, except in high mountain valleys; and hence we have, erroneously I think, been led to add “cold” to the other qualities of the climate best suited for the tuberculous.

I am satisfied that the sanatoria in the Swiss Alps but very imperfectly supply the ideal climatic requirements of the phthisical, and scarcely present the best conditions for the cure of phthisis generally. I say generally because I know they suit a certain special class of consumptives extremely well.

In the first place, the atmosphere of these sanatoria is by no means always unirritating. On the contrary, it can be at times

* *Op. cit.*, p. 379.

excessively irritating. The extreme variability of different seasons and of climatic and meteorological conditions, even in the same season, are distinctly injurious to many cases, and have certainly proved fatal to some who would not have succumbed in a less rigorous and irritating climate. Attacks of acute pleurisy, pneumonia, and bronchitis are in some seasons of not infrequent occurrence. My own personal experience may be exceptional, but I have not seen such good results from a residence in the Swiss Alps, especially in serious, well-characterised cases, as I have from the climate of South Africa or that of South California; or in more advanced cases from Madeira or the Canaries; and I could, at this moment, put my hand on cases that show better results from residence in some of our own south coast resorts than I could from residence in Switzerland.

I believe, however, that the quite early pre-phthisical tuberculous cases, and even more advanced cases in vigorous organisms, do well in these resorts. Certain neurotic cases seem also to do extremely well in the Swiss Alps, mountain air appearing to have a sort of fascination for them.

But I have tested these climates with cases of decided and well-developed disease, and of such cases I can point to very few survivors, and I know this has been the experience also of non-medical and non-phthisical frequenters of these resorts. Of the phthisical acquaintances they have made there, few, they tell me, survive. I have seen much more remarkable results in the way of prolonging life in serious cases from the climate of Madeira, where living in the open air is possible for many more days in the year and many more hours in the day, and the air is soothing and not irritating.

Next to life in the open air—or better, associated with it—there is another important condition of cure, and that is continuous and strict attention to minute details of daily life and hygiene. With such a condition rigorously carried out even an indifferent climate may be turned to good account. *Im Kleinen grosse*—great in small things—such is the device of Dr. Dettweiler, who has conducted his now well-known sanatorium for phthisical patients at Falkenstein with so much success. It is situated close to Frankfort-on-Maine, in the cold damp climate of the Taunus hills—by no means such as we should think well suited to the treatment of phthisis. But minute attention to the smallest details—for nothing is too

small or insignificant for Dr. Dettweiler's personal direction—and absolute obedience to instructions are rigorously insisted on. Air, food, exercise; not, however, excluding medicinal treatment—these are the curative agents on which he mainly relies.

Out of 1,022 cases of undoubted phthisis, treated at Falkenstein between 1876 and 1886, 132 left quite cured and 110 relatively cured. These are the results obtained in what might be called a bad climate.

There is another therapeutic agent which is almost universally esteemed as influential in promoting curative changes in tuberculous deposits in the lungs in the early stages, and that is counter-irritation—repeated and continuous counter-irritation.

With regard to the use of chemical and pharmaceutical agents I have not much to say. The great divergence of opinion that exists, and has existed, with respect to the value of the various agents of this kind that have, from time to time, been advocated as curative of phthisis may, to some extent, be explained by reference to the varying conditions under which they have been applied. It has seemed to me, after many years of careful observation, that the tissues of one individual will react to a remedy in a very different manner to the tissues of another; just, indeed, as they differ in their reactions to the infective morbid agent itself. And the results are different because the conditions, though undiscoverable, are different. So a particular agent or remedy may confer upon a particular tissue a resisting power it would fail to confer on another. This is the way in which I have explained to myself the remarkable differences in the action of different remedial agents on different individuals.

I have, like others, from time to time, gained some excellent results from the hypophosphite of lime, and at one time I used it very largely in a great number of cases of phthisis. I use it less now, and with more discrimination, because in many cases it has seemed to me to be utterly useless. It is far more useful in the cases of children and young people than in adults, and in old people I believe it to be inert. It is far more effective in florid fair people than in dark, sallow ones. The latter, by the way—the very dark, sallow, tuberculous subject—is the most difficult of all to produce any remedial effect upon by drugs or, for that matter, by any other therapeutic process.

The introduction a year or two ago of the Bergeon method of

treating phthisis—the introduction of sulphuretted hydrogen gas into the large intestine—will be remembered. It is rarely or never used now, and yet I saw some excellent results from its application in a limited number of cases; and I remember that my friend Dr. Coghill, with his quite exceptional opportunities at Ventnor of observing the effects of this and other methods in the treatment of phthisis, spoke of it as the most promising he had ever tried. I wonder if he ever uses it now. Dr. Henry Bennet, of Mentone, spoke also enthusiastically of its curative effects.

I had also some good results from the use of tuberculin, and except in one case I have personally encountered no unfavourable results—that was one of those dark, swarthy, extremely cachectic subjects—a type of consumption I have never known to respond favourably to any kind of treatment. One small injection developed in this case the signs of a widely diffused pulmonary infiltration, which we suspected but had not been able to clearly ascertain before the injection of tuberculin. I no longer use it because the alarmist reports in the newspapers and the private denunciation of so many medical men have made patients indisposed to submit to it. Certainly the manner in which this remedy was introduced was deplorable, and the irritation it excited amongst members of the profession in this country and elsewhere was perhaps not to be wondered at. Still it would have been better if less feeling had been shown in its denunciation. I think it will survive in a modified form the temporary opposition to it.

Certain antiseptic agents I believe to be of great value in influencing favourably the course of pulmonary phthisis. I have hardly seen a phthisical patient who has diligently and adequately used antiseptic inhalations who has not derived considerable, and in many cases lasting, benefit from their use. But in this as in so many other things, it is remarkable how few patients can be induced to persevere regularly and adequately with inhalations; and, again, their beneficial influence is much more remarkable in some cases than in others. I find, however, in phthisical patients that I have lost sight of for years, when they return they almost invariably refer the improvement they have experienced to the persevering use of antiseptic inhalations.

Of antiseptics given internally I have seen none so uniformly beneficial as creasote or guaiacol. It has held its ground better than any other antiseptic agent that has been applied to the treat-

ment of phthisis; it has been in use for many years, and may, indeed, be looked upon as a descendant of the very old tar treatment. It has steadily gained in favour, and it is now, perhaps, more widely used, in every country in the world, than any other remedy for phthisis. Quite recently a method of administering it in large doses by the bowel has been warmly advocated.

But in the use of any remedy for consumption, in order that it may cure or contribute to the cure of this disease, the conditions of cure must exist. No remedy can cure incurable conditions. And I repeat now what I have already said, that if we knew at this moment of a certain cure for tuberculosis the majority of cases of pulmonary consumption as they now exist would remain incurable, because the conditions of cure, although they may have existed at one time, exist no longer.

The PRESIDENT said that every pathologist must have seen evidences of extinct tubercular disease, and these extinct lesions often broke out again many years later after catarrh or congestion; the bacilli apparently finding under these conditions an opportunity of again developing in the increased quantity of plasma produced by the congestion. We had no ready means of proving the presence of spores in extinct lesions, but this could be ascertained by injection into animals. The great condition necessary for cure was a limited lesion. He thought that the division which the author had made between tuberculosis and phthisis was purely arbitrary; it was merely one of degree. Given a certain extent of phthisis, the question of cure depended upon whether the patient could pass through the necessary stage of elimination of caseous material. The efficacy of the treatment by tuberculin depended upon the fact that it hastened the eliminative process; it was undoubtedly a two-edged weapon, and we required more experience before we could with propriety use it in any considerable number of cases. He quoted a case in which advanced phthisis was arrested under the Weir-Mitchell treatment. Creasote and guaiacol were useful remedies, but they required decided pushing.

Dr. DE HAVILLAND HALL emphasised the necessity of adapting the details of treatment to the needs of each individual patient, and he quoted a case to illustrate this. The patient, whose place of business was in a low-lying and damp locality, had been ordered abroad on account of incipient phthisis. As he was unable to carry out this recommendation, he consulted Dr. Hall, who advised him to live away from his place of business, on high and dry ground. The advice answered very well, as the gentleman was able to continue his occupation, and the physical signs of active mischief gradually cleared up. The treatment adopted in this case consisted of energetic counter-irritation to the chest by tincture of iodine, inhalations of creasote, copaiba capsules when the expectoration was profuse, and tonics. Dr. Hall highly commended the perforated zinc inhaler devised by Dr. Yeo.

Dr. F. L. BENHAM said that the use of sulphuretted waters had been recommended in the treatment of phthisis in the writings of Dr. Armstrong; but the systematic resort to this method of treatment seemed

now to be completely abandoned in England, though it was still employed to some extent in France. Yet if benefit resulted from Bergeon's treatment, it seems likely that equal success would follow the use of sulphuretted water in appropriate cases; and this method would be simpler and less disagreeable than the injection of sulphuretted hydrogen into the bowel.

Dr. C. D. F. PHILLIPS had found that patients in the last stage of phthisis were not benefited by Madeira; no island for its size had so much phthisis among its inhabitants. He thought too much had been said in praise of creasote, which, however, might be of great value where there was a gangrenous tendency.

Dr. YEO, in reply, said that the distinction he drew between tuberculosis and phthisis was merely a clinical one. He agreed that the effect of tuberculin was to hasten elimination. He thought that nearly all the therapeutic remedies he had mentioned acted by diminishing irritability of tissue. The French rarely resorted to forced feeding with the pump now, but they largely used a preparation of beefsteak in powder. Cases of phthisis treated in bad surroundings presented one constantly with surprises. In the Pyrenees were two health resorts with sulphuretted hydrogen waters, which were claimed to produce much benefit in cases of phthisis. He quoted the case of a patient with advanced phthisis whose life had been prolonged for six years by residence in Madeira, which was now much more popular than it was ten years ago. He repeated that he had seen better results from creasote than from any other remedy; it improved the appetite, diminished catarrh of the lung, and he thought it favoured sclerotic changes.

November 16th, 1891.

TWO CASES OF PERFORATING ULCER OF THE DUODENUM IN WHICH EXPLORATORY LAPAROTOMY WAS PERFORMED.

By C. B. LOCKWOOD, F.R.C.S.

PERFORATING ulcers of the duodenum are so unusual that the occurrence of two in the practice of one surgeon within a period of three years is, perhaps, worthy of being recorded. Unfortunately these cases throw no new light upon the pathology of this ill-understood disease, nor do they afford indications for its more successful treatment. On the other hand, they are typical instances of their kind, and it is not impossible that when they have been collated with others they may help to afford materials for the diagnosis of cases which may be met with in the future.

The first case in which laparotomy was performed for septic peritonitis due to a perforating ulcer of the duodenum was that of

a young man aged 28 years, a clerk, who was sent into the Great Northern Central Hospital under my care by Dr. Rout. He was admitted on July 2nd, 1888, with the following history. He had always enjoyed excellent health until four days before admission, when whilst drinking a cup of tea he was suddenly seized with a violent pain in the left side. This was thought by Dr. Rout to be an attack of colic; but the next day it is said that there was a well-defined tumour to the right of the umbilicus, and the patient was sick and constipated. These two symptoms continued, and the abdomen became distended and tympanitic, and the tumour became obscured. Towards the end of the second day the vomit became green, sour, and afterwards stercoraceous. There was never any history of blood, either in the vomit or in the motions. On the third day Dr. Cowie, our house surgeon, ascertained that the patient had complete obstruction, with a pulse of 130 and a normal temperature. His expression was very anxious, and there was great distension of the abdomen. I could find no particular area of dulness; there was no vermicular movement in any of the intestinal coils, which could be plainly seen through the thin abdominal walls. With the view of clearing up the question of a mechanical obstruction, the abdomen was opened by an incision along the linea alba to the left of the umbilicus. This gave exit to a quantity of foul-smelling gas, and there was general purulent peritonitis, the pelvis being almost full of pus. The whole of the intestines were carefully examined, but no perforation was discovered. The distended intestines were relieved by a small incision, which was afterwards closed by Lembert's suture. The opening and emptying of the intestine were followed by great collapse. The abdominal wound was closed and drainage established in the usual way, and, although the patient had no more pain, he died seven hours after the operation. At the examination the usual signs of general septic peritonitis were found. The chief focus of this was beneath the liver, and upon the front surface of the duodenum there was a small oblong aperture, which gave exit to the duodenal contents. The margins of this aperture were clean cut, and the mucous membrane in its vicinity was thin and congested. There is no doubt that this perforation was the source of the septic peritonitis.

The second case was that of a man aged 41, who was admitted under my care in May, 1891. He seems to have been a

healthy man, except that a year before he had had an attack of abdominal pain, accompanied by constipation; also he seems to have suffered slightly from indigestion. His present illness began suddenly. Whilst at work he was seized with a violent pain in the abdomen, followed by complete constipation and by vomiting. The vomit at first consisted of the contents of the stomach, but afterwards became stercoraceous. There was never any evidence of blood either in the vomit or in the motions. When first seen his expression was anxious, and he complained bitterly of the pain in his abdomen, which was rather tightly distended and tympanitic in every part, except the flanks. The contour of the abdomen was regular throughout, and no respiratory or vermicular movements could be detected. There was nothing in the rectum or in any of the hernial apertures to explain the symptoms of the obstruction. His pulse was rapid, about 120 per minute; and although his temperature was only 99·2° F., I diagnosed diffuse septic peritonitis. However, aware of the uncertainty of such a diagnosis, and fearing lest a mechanical obstruction might have been overlooked, the abdomen was explored through the usual incision. Diffuse septic peritonitis was found; but although every region was searched, nothing could be found to account for its occurrence. The abdominal cavity was washed out and drained, and closed with complete assurance that no mechanical obstruction existed. The unfortunate man died ten hours after this operation, and at the examination, which was made by Dr. Gallo-way, the peritoneal cavity was found intensely inflamed, with lymph between the coils of intestines and collections of purulent fluid. This condition was most intense beneath the liver, in the region of the duodenum, which latter was perforated by a small circular ulcer. This ulcer had sloping edges, and was situated at the hinder part of the duodenum, about an inch from the pylorus. It was about half an inch in diameter where it opened into the bowel, but smaller towards the peritoneum. It gave free exit to the duodenal contents when the bowels were lifted up. Nothing else which seemed noteworthy was ascertained.

Laparotomy was performed in these cases because, although it was strongly suspected that the symptoms were due to septic peritonitis, it was feared that there might have been a mechanical obstruction. With our present knowledge it is, I fear, impossible to discriminate with certainty betwixt intestinal obstruction due

to mechanical causes and that caused by diffuse septic peritonitis. It would have been a great misfortune to have overlooked a mechanical obstruction, which might perhaps have been easily remedied. The operation decided the non-existence of a mechanical obstruction, although in neither case was the actual cause of the peritonitis discovered. It looks, at first sight, a simple matter to explore the abdomen and ascertain that there is not a mechanical obstruction, but the task is more difficult than it seems. If, when the abdomen has been opened, collapsed intestine is seen, it is of course a clue to the point of obstruction; but in these cases none existed, and therefore the usual procedure was followed and the cæcal region at once explored. Beginning thence, it was easy to ascertain that the whole of the large intestines, as well as the small, were uniformly and tightly distended, and that there was also general septic peritonitis. To bring into view the perforation in the first case, the abdominal incision would have had to have been prolonged almost to the ensiform cartilage; and in the second case the aperture was behind the duodenum, in an almost inaccessible position. Beyond this the operation calls for nothing further except to remark that the intestinal distension is an important factor in these cases; it impedes the abdominal exploration, and there is hardly any chance of success if the abdomen is closed without its having been relieved. In the first case this was done by making a small incision into the intestine, which was afterwards repaired with Lembert's suture; in the second this step was not required. Although the diagnosis of diffuse septic peritonitis is exceedingly difficult, yet I am beginning to believe that the task is not impossible. The symptoms are those of acute intestinal obstruction, and often, as in the cases I have just described, the absence of premonitory symptoms, the sudden onset, and the absence of any rise of temperature, leave few indications for a correct diagnosis. However there are other signs worthy of being considered. It is a question whether the intestinal obstruction is ever complete. I have never seen a case of peritonitis in which there was not some passage of flatus. Next, the vomit is sour or bilious in peritonitis, and does not become stercoraceous until many hours have elapsed; sometimes it is never quite stercoraceous. The pain, too, is often described by the patient as being of a burning character, and, if at first localised, soon becomes general throughout the abdomen; there is, as a rule, at this stage, pain

on pressure in every region. The abdominal distension, too, is perfectly regular, and, in the cases I have examined, the percussion note is clear everywhere, both over the small intestines and over the cæcum, and along the course of the large intestines. There is one other sign upon which, I believe, great stress ought to be laid. Unless the patient is stout, the coils of the distended intestines can usually be discerned through the abdominal wall. Now in mechanical obstruction these coils exhibit vigorous peristaltic movements until their muscular walls become exhausted; in peritonitis the peristaltic movements are abolished as soon as the inflammation is established. I have not mentioned the negative aids to a differential diagnosis, but the importance of a rectal examination, of blood or mucus in the motions, of tumours or indurations in the region of the cæcum or sigmoid flexure, and of the numerous indications afforded by the age and sex of the patients and by the previous history, are too obvious to need pointing out. The pathology of these ulcers of the duodenum is in such a doubtful and uncertain position that I do not propose to attempt the question. It is strange that they should be so much commoner in men of about thirty years of age than in women.

I must express my indebtedness to Dr. Cowie for the notes of the first case, and to Mr. Falkner for those of the second.

The PRESIDENT asked if the temperature was depressed below the normal. It was remarkable how intense a peritonitis could exist with a subnormal temperature. He could not agree with the authors in their disposition to operate so early before the diagnosis could be well established. In addition to the signs which had been alluded to as aiding in the distinction between perforative peritonitis and mechanical obstruction, it should be remembered that in the former the normal hepatic dulness often entirely disappeared.

Mr. JESSETT congratulated the author on having brought these two very rare cases before the Fellows of this Society. That they were rare was testified by the fact that in fifteen years Dr. Norman Moore had only found two cases recorded at St. Bartholomew's Hospital. Mr. Jessett quite agreed with the line of treatment adopted by Mr. Lockwood, as there could be no doubt if operative interferences were to be of any avail they should be had recourse to early, and if suppurative peritonitis had set in the abdominal cavity should be flushed out with warm water and drainage adopted. Mr. Lockwood's cases were the more puzzling as on opening the abdomen the intestines were all found greatly distended. Now in mechanical obstruction, if on the cæcum being sought for it was found to be distended, the surgeon looked for the seat of obstruction below that point, usually the sigmoid flexure or rectum, if the cæcum was empty the seat of obstruction would be found in the small intestine. In the cases before them there was no obstruction,

and Mr. Lockwood adopted the wisest course he could in making a free incision into the intestine and emptying it. In these cases a small incision was useless as the intestine was paralysed and lost all power of contractility, the intestines instead of contracting as a healthy intestine did, simply collapsed, and in these cases it was found necessary to empty the intestines by lifting loop after loop, and let the contents escape by gravitation. Mr. Lockwood, after careful examination, found neither obstruction nor perforation, although he examined the small gut most carefully. Possibly had he torn through the lesser omentum or the gastro-colic omentum he might have discovered the point of leakage and been able to stitch it up, but seeing the great rarity of these ulcers of the duodenum, one can easily imagine that such a course was not deemed necessary, but with these two highly interesting and instructive cases before us, I cannot but think that if a similar condition of things was found in another case the surgeon would be wise to tear through one or both of the omenta and examine the duodenum carefully for the perforation. Time will not permit of my here more than suggesting that a good method of finding these ulcers is to force hydrogen gas or compressed air into the gastro-intestinal tract; this can well be done through the opening made for the emptying of the intestine.

Mr. CRIPPS, referring to the question of diagnosis, said that a sudden onset of pain usually indicated the presence of mechanical obstruction, whereas it came on more gradually in perforation. It was an important point to decide how long after a perforation one could wash out the cavity of the abdomen with prospect of preventing peritonitis. He alluded to a case of inguinal colotomy which had been under his care. All was going well, and on the sixth day after the operation a dose of castor oil was administered. Half an hour after a fit of coughing the patient complained of pain, which in the course of an hour became intense in its severity. When he saw her some time later he found that the opened intestine had given way and had dropped back into the peritoneum, and that after this had occurred a copious motion, the result of the action of the castor oil, had taken place into the peritoneal cavity. He enlarged the wound and thoroughly washed out the peritoneum, re-stitched the gut to the belly-wall, and put in a drainage-tube. Almost from the moment of the flushing out the pain began to subside, no peritonitis developed, the patient recovered, and remained well a year and a half afterwards. From a study of this case two points were clear: that pain might be produced immediately by extravasation, and that faecal matter might remain in the peritoneum for three or four hours, and then be washed out without peritonitis developing.

Mr. LOCKWOOD, in reply, said the temperature was neither depressed nor elevated. He adhered to his opinion that the time to operate was early, before distension had set in, although he was ready to admit that there was a time which was too soon, when diagnosis was doubtful. If there were great distension of the gut with air, the liver dulness would diminish in an upward direction. The incision which he made into the small intestine was an inch in length, and was quite adequate to give the relief required. He felt sure that puncturing was not a course which many surgeons would adopt, though one could not forget the brilliant results which had been obtained by Dr. Oliver of Harrogate, by which he had apparently cured some cases of chronic intestinal obstruction. If he met with another such case he would probably explore the duodenum, and he admitted that there would be no difficulty in adequately suturing the ulcer if one should be found.

ON AMPUTATION OF THE HIP-JOINT, WITH RECORD OF TEN CASES.

By RICHARD DAVY, F.R.C.S. Eng., F.R.S.E.

FOR fifteen years I have been most interested clinically in cases of hip-joint disease that demand surgical severity. I trust that a summary of my experience may have a practical value, and may render somewhat less the anxiety and responsibility of surgeons undertaking this the gravest amputation of all, viz., amputation at the hip-joint. The gravity of such procedure ought to forcibly impel surgeons to take measures to prevent its necessity. Early diagnosis of ilio-femoral disease, intelligent treatment, and perseverance with general and local measures should be written in bold type on the title-page of its history. Yet, in spite of warning, care, and weariness of well-doing, cases eventuate whose only solution rests in ablation of the joint or of the limb. Traumatism plays an important initial part; insanitation and poverty supplement it; an amputation at the hip-joint amongst the well-to-do classes is almost unknown, bearing out the accepted dogma, that "circumstances alter cases." Let us take an instance where continued and exhaustive suppuration, hectic, hill and dale on the temperature chart, emaciation or commencing lardaceous disease of the viscera compel interference; then as a rule it is better to excise the joint in view of giving a patient the chance of improving, and as a preliminary step in any future operation. Yet personally I do not view excision of the hip-joint very favourably in any but young subjects; children up to and below ten do admirably after excision of the hip; over that age the dangers are greater, and the ultimate usefulness of the limb not so good.

The record of my own personal experience shows that I have operated on ten patients in fourteen years, all except one have been markedly inveterate cases of morbus coxæ; four of them had suffered excision prior to amputation at the hip-joint, profuse suppuration and resultant enfeeblement have been the immediate conditions necessitating amputation; all these doubly operated upon patients have recovered, one good argument in favour of subdividing an amputation at the hip-joint into two stages, as advocated by Keetley; the mortality is 20 per cent; both were

Ten Cases of Amputation at the Hip-joint.

No.	Sex.	Age.	Date of admission.	Nature of disease.	Operation.	Departure.	Result.	Blood lost.
1	Boy	9	July 10th, 1876	Morbus coxæ (right)	Jan. 16th, 1877	May 16th, 1877	Recovery	1½ oz.
2	Boy	5	Aug. 14th, 1878	Morbus coxæ (right). Excision previously	July 16th, 1879	Oct. 21st, 1879	Recovery*	1 "
3	Boy	8	Nov. 28th, 1879	Caries. Left dislocation. Sequel of scarlet fever	Jan. 20th, 1880	June 24th, 1880	Recovery	5 "
4	Man	29	June 30th, 1880	Pelvic necrosis (left)	Oct. 15th, 1880	Oct. 15th, 1880	Death from shock in 5 hours	4 "
5	Boy	16	April 18th, 1882	Excision, July 11th, 1882 (right)	Nov. 21st, 1882	Feb. 24th, 1883	Recovery†	1½ "
6	Girl	18	Oct. 4th, 1885	Old dislocation at 2 years old (left morbus coxæ)	Nov. 13th, 1883	March 28th, 1884	Recovery	2 "
7	Girl	23	Jan. 16th, 1885	Excision of right hip-joint, Jan. 22nd, 1883. Flail limb	Jan. 27th, 1885	May 1st, 1885	Recovery	3 "
8	Man	43	May 4th, 1886	Necrosis of left head, neck, and shaft of femur	June 8th, 1886	June 9th, 1886	Death from syncope (18 hours)	2 "
9	Boy	10	Oct. 21st, 1886	Morbus coxæ (left)	Oct. 29th, 1886	Jan. 25th, 1887	Recovery	2 "
10	Boy	14	Oct. 20th, 1890	Morbus coxæ (left) Excision, Nov. 9th, 1890 (elastic tourniquet)	Jan. 20th, 1891	April 21st, 1891	Recovery	2½ "

* Boy died May 23rd, 1890.

† Ambulanced to Harlington. Death in the Royal Free Hospital from pelvic necrosis on Feb. 6th, 1890.

men, aged 29 and 43 respectively. Two of the patients have subsequently died, one eleven, the other eight years after operation. The first of these (Case 2 on the table) was supposed to have had lardaceous disease of his viscera, at any rate he had copious albuminuria and epithelial casts in his urine in 1878. In speaking of this boy in 1880 it was remarked: "This boy had lardaceous disease of his viscera, with distressing ascites. The rectal lever was used. It would have been impossible to compress his aorta or common iliac artery through the abdominal wall, by reason of the distension. This boy's recovery is pleasing, and the albumen in his urine is daily diminishing." On April 7th, 1890, he was readmitted into Westminster Hospital from Dr. Barnardo's Home, the subject of urgent and uncontrollable sickness, with a puffy and discharging right stump of thigh, and an œdematous foot (left). His urine was albuminous, alkaline; sp. gr. 1016. The patient passed about 11 oz. of urine in twenty-four hours, and there was persistent diarrhœa. On May 1st a piece of bone was removed from the stump and sinuses were laid open. He got weaker, duller, and more helpless, dying on May 23rd, 1890. Appended are Dr. Hebb's notes of the necropsy. "These I need not state fully, but will give an excerpt. The kidneys weighed $9\frac{1}{2}$ oz. and $9\frac{1}{4}$ oz. right and left respectively. They are typical examples of large white kidney. None of the viscera present any of the characteristics of lardaceous disease, nor do they give any amyloid reaction with iodine." We see, therefore, how difficult it is to diagnose lardaceous disease in these exhausted surgical cases. Case 5 died on February 6th, 1890, eight years after operation, in the Royal Free Hospital, of pelvic necrosis. I positively declined to see the complete *post-mortem* examination on this boy's body because the *post-mortem* room was filled with women. In my opinion women are quite out of place in the deadhouse of any general hospital. So, as they would not leave, I did.

Method of operating.—In 1880 (Surgical Lectures) I stated: "With reference to the method of operating, I shall only advise you to exercise common sense, and apply it to each individual case; keeping the skin intact where the scrotum rubs the thigh, saving as much soft tissue as you possibly can, and having your front flap covering the wound epaulette fashion, for the production of a dependent drainage, and the utmost simplicity of future dressing." Furneaux Jordan, in recapitulating the essentials of his operation,

says, "I conclude by again repeating the principle of the operation—enucleate the bone where it is most thinly covered; cut across the soft parts where they are smallest; do not touch the bulby soft parts at the inner and upper parts of the thigh."

Professor Ollier, of Lyons, did this operation in 1859, but the credit of bringing the method prominently before surgeons is, in my opinion, greatly due to Furneaux Jordan. The lever or elastic tourniquet having been applied, a circular incision is made at about the upper third of the thigh, through all the soft structures down to the femur; the lower section is drawn down forcibly towards the knee, to give space for the ligation or torsion of vessels. The bone is not divided, but kept intact for future leverage in disarticulating at the hip-joint (a most valuable act of conservatism). After all vessels are carefully secured, an external wound is made right down to the shaft of the femur; the muscles around the trochanter major and the shaft of the bone are freed from their attachments; utilising the under surface of the rectus femoris as a guide, divide the psoas and iliacus on the capsule of the joint, as well as the capsule itself, by a long anterior slit and two short crucial nicks. The assistant then rotates the limb outwards, and forcibly adducts the knee towards the opposite armpit to dislocate the femur from the pelvis. By the yet further availability of the limb and femur as a lever the member is removed by a few well-directed touches of the knife, any vessels which may have been injured are promptly secured, the acetabulum is scraped, if necessary, the wound is thoroughly syringed with hot water and corrosive sublimate solution, stitches are put in, and the patient is at once conveyed to a warm bed. Use all proper speed in operating, short of hurry. "*Festina lente*" is a good motto for convenient haste. Only one word about the lever. Nine out of these ten cases have submitted to its employment. The elastic bandage in the hands of a competent assistant is quite sufficient to control any hæmorrhage, and especially so in those cases where excision of the joint has preceded amputation. The elastic cord must be applied obliquely, spica fashion, and be manipulated so as not to interfere with abdominal respiration. The use of the elastic cord is absolutely free from danger; that of the lever is not.

Let me correct an error that has frequently been expressed, that an amputation at the hip-joint involves a loss of nearly one-fourth of the human frame. I have already contradicted this and can

adduce chapter and verse on this point. On January 20th, 1891, I amputated at the hip-joint of a boy aged 14. The weight of the parts removed was 4 lb. On February 19th, 1891, I weighed the boy—as soon as I felt it safe—and his body weighed 50 lb. Admitting that he might have gained 2 lb. in weight since the operation, we have the proportion of tissue removed to tissue saved as 4 to 48. This reduces the mutilation to one-thirteenth of the boy's body—a great difference from one-fourth. The point of selection (upper third of thigh) as now practised necessarily makes the comparison favourable; for in olden days the catlin entered at the anterior superior spine of the ilium, and emerged near the tuber ischii, slap-dashing off the limb by two flaps.

On October 17th, 1881, the late Mr. Shuter amputated a boy's left thigh at the hip-joint* by a subperiosteal method. In 1882 the boy had a strong movable stump, and it had the appearance of an amputation in the middle third of the thigh. An artificial limb was made for the boy by Mr. Gray. This was worn for five months, and it had to be discontinued by reason of a painful sinus, which opened opposite the acetabulum. The case was exhibited on February 9th, 1883. The committee appointed to report on the condition of the boy's stump state that by “the preservation of the muscles of such a length, and with their attachments becoming so firmly united around a strong central cord, the result obtained approaches that of an ordinary amputation at the upper third of the thigh, in respect both to the facility of adapting an artificial limb and the power with which the patient can move this appliance when walking.”

My patients are all walking with the aid of crutches, except one, who has an artificial limb. For working folk crutches are such a support, such a warning to outsiders, so easily discarded or resumed, and so comparatively cheap, that practically they are preferred in cases of this nature.

The explanation of the reduced mortality from 60 to 20 per cent. appears to be a general one.

1. The use of anæsthetics. We are too apt to forget the benefits handed down to us by previous workers better than ourselves, and to pocket their results as if they were our own. The patient being free from pain, permits a more careful and studious dexterity on the part of the surgeon, and lessens the shock on the part of

* *Vide* ‘Clin. Soc. Trans.,’ vol. xvi, p. 86.

himself. 2. The more rigorous application of measures calculated to prevent the loss of blood; such as the lever, elastic bandage, torsion, and the composed conduct of dressers. 3. The diminished shock, by adopting the method of operating at the juncture of the upper and middle third of the thigh instead of the old high transfixion plan; the less risk of hæmorrhage (primary and secondary); and the uninjured condition of parts at the inner side of the thigh, situated in the confines of Scarpa's triangle. 4. Improved sanitation, nursing, and hygienic surroundings; more skilful dressings; marked cleanliness, and the use of the syringe. 5. The greater quiet of isolation, the careful selection of cases for operation, and the better sustained discipline preparatory to and after the operation.

Let me again advocate every possible trial on the part of the surgeon to prevent the necessity of recourse to amputation at the hip-joint. Should such necessity occur as a last resource for saving life, let the operation be done on somewhat similar lines to what has been narrated in this paper; let it be completed with resolution and composure. Then, after the daily and nightly care of surgeon and nurse for, maybe, weeks or months, the patient will be able to walk on crutches or with the aid of an artificial limb, and enjoy a further lease of life.

Mr. STEPHEN PAGET referred to two cases of amputation at the hip-joint: in one of which, being unable to do Mr. Furneaux Jordan's operation, he had first tied the femoral before cutting the flaps.

Mr. LOCKWOOD had not gathered from Mr. Davy's remarks what opinion he had formed of subperiosteal amputation. Cases had been mentioned, but without comment. The removal of the femur from within the periosteum made the operation so much easier, that the temptation to take that step was very great. However, some years ago Mr. Lockwood had seen a case of subperiosteal amputation at the hip in which the periosteum threw out no bone, and remained a suppurating cavity, which caused the death of the patient from amyloid disease. Also, in another case the periosteum, after having been left behind, threw out a quantity of ill-formed and unserviceable bone, which was afterwards operated upon with a view to its removal. Evidently subperiosteal amputation at the hip should not be undertaken without much consideration.

Mr. DAVY, in reply, said that he would treat the early cases in the way now usually adopted. In all the cases in which he had operated, everything else had been tried, such as rest, splintage, and cod-liver oil. He confessed that the case in which he had diagnosed lardaceous disease to be present was not found to be so after death. He had no experience with intravenous injections. In a case of lardaceous disease he would certainly perform amputation in order to try and save the patient's life. In Mr. Shuter's case the result was very good, and one advantage of the subperiosteal method was that it promoted free drainage of the structures of the thigh.

November 30th, 1891.

ON A HITHERTO UNDESCRIBED FORM OF EPI- DEMIC SKIN DISEASE.

By THOMAS D. SAVILL, M.D. Lond., D.P.H. Camb.*

IT is admitted by good observers that the form taken by minute fungi and by bacteria is greatly influenced, not only by the source of the spore, but by the medium in which it is grown; and that spores of the same species, vegetating under different conditions, develop such differences of form that it is impossible to recognise them as being related one to the other.

Out of an average of some 500 patients in the new infirmary and infirm wards of the workhouse adjacent, it is rarely that we are without half a dozen cases of more or less localised eczema; not unfrequently cases of acute general eczema come in, and occasionally a case of pityriasis rubra is admitted. Such cases are specially liable to occur in the autumn, so far as my memory serves me; but never before have we had an epidemic of skin disease at all approaching the present one. Out of 846 (376 males, 470 females) patients who passed under treatment in the infirmary and workhouse infirm wards between July 1st and October 31st, 163 (89 males and 74 females), or 19·2 per cent., have been attacked with the disease I am about to describe, up to the present time.† Some had the disease very trivially, but most had quite half the surface of their bodies attacked, and many were entirely covered with it. In a few it scarcely interfered with their usual condition, but in most the irritation of the skin and the concurrent constitutional disturbance rendered their condition a very unhappy one, and 28 of those attacked died.

It may be mentioned that the disease broke out not only in many different wards at the same time, but also in two buildings

* Numerous cases, photographs, and microscopic specimens were exhibited. Notes of many of the cases are published, with illustrations, in the *British Journal of Dermatology* of February, March, and April, 1892, reprints of which have been published by H. K. Lewis, Gower Street.

† When I first drew attention to the disease, I took it for acute general eczema, but as day after day fresh cases occurred, I could not help noticing what a marked difference there was between a typical case of this complaint and eczema in any form.

which are totally distinct.* The cases also were pretty evenly distributed, excepting in the nursery and children's ward, which contained very few cases, and those chiefly among the adult patients who happened to be there at the time.

It is very important to observe that 152 of the cases occurred amongst the persons already inmates of the infirmary or workhouse for some other disease. Only one man (M. 56)† was brought in with the skin affection upon him, at a date when many other patients were already affected.

Only five male and six female cases occurred amongst the able-bodied or aged inmates of the workhouse.

Amongst the paid staff of either place there was remarkable immunity, the only ones attacked being myself and a housemaid; my little dog was also attacked. These three instances are not included in the tabular statements.

All the 163 cases, though differing considerably in detail, bear a strong resemblance to each other, and, whatever may be the explanation of the epidemic, it will, I doubt not, be conceded by the many gentlemen who were kind enough to see them that they are all examples of one disease, one pathological entity. That they vary amongst themselves is as true of this as of any other disease.

They presented differences both of kind and severity just as do cases of localised eczema or any other disease.

Description of Illustration.

CASE F. XXI.—Maria T—, aged 68, the subject of our illustration, *naturally a thin woman*, was admitted to No. II ward on October 29th, 1890, for an eczematous ulcer of the left leg. The patient had formerly had "eczema," and, six years before, an attack of erysipelas, followed by abscesses and loss of hair. The ulcer took nearly a year to heal; and it left a patch of chronic eczema on the left leg and thigh, which persisted until after the epidemic attack, *and then disappeared*.

On August 6th the epidemic malady started, not on the leg, but as *a clearly defined oval ring under the chin, perfectly clear in the centre, red and*

* The Paddington Infirmary, like the Marylebone Infirmary, where a similar outbreak occurred at the same time, is a modern building, replete with all the latest sanitary improvements, administered under the medical superintendent. The infirm wards are a part of the workhouse, and governed by the master. The staffs of the two places are totally separate, and the buildings, though adjacent, stand in different grounds, separated by a wall. The only points they have in common are the medical staff, who have charge of both institutions, and the board of guardians, which governs both.

† Reference numbers of the cases, the notes of which are open to inspection.

EPIDEMIC SKIN DISEASE (SAVILL)



CASE F. XXI. (naturally a thin faced woman).—Three weeks from real commencement. Shows swelling of arms and face, which was so great as to close the eyes at one time.

raised at the margin, the size of half a crown. This, after spreading a little, faded away in the course of a week. Then the eruption *broke out with redoubled vigour* on the forehead, and rapidly spread. This time the eruption took the form of discrete papules, such as are seen on the chest in the illustration, which soon become vesicular. This phototype shows the swelling and thickening of the skin round the neck and eyelids, so great that the latter could not at one time be opened. The whole body was attacked, sooner or later, but the arms and face were always the worst, and here the exudation was considerable. The face at one time was half again as big as natural. The primary attack lasted $6\frac{1}{2}$ weeks, and was followed by a slight relapse. The temperature varied between 97° and 99° ; once it reached 100° , when the disease was at its height.

The anorexia and asthenia were marked, at one time recovery being despaired of; there was some albuminuria, and the rash was followed by general wasting, alopecia, and leucoderma. *The initial patch beneath the chin is still marked by a white area, which contrasts strongly with the natural colour of the skin around.*

DEFINITION.

As a provisional definition I have adopted the following:—

A contagious malady in which the main lesion is a more or less universal dermatitis, sometimes attended by the formation of vesicles; always resulting in extensive desquamation of the cuticle; accompanied by a certain amount of constitutional disturbance, and running a more or less definite course of seven or eight weeks. With a view to arrive at an average on the various clinical features I have, with the help of my colleagues, prepared several tables, which I am able to present to you to-night.

THE SKIN LESION.

For purposes of description the eruption is best divided into three stages:—

1. *Papulo-erythematous Stage* (lasting 3—8 days).—(a) The eruption usually commenced as a discrete papular rash, such as may be seen on the chest in the photograph of Case F. 21 (*vide* illustration).

(b) Nearly all the serious cases began as an erythemato-papular rash, *i.e.*, a congested surface on which numerous small shotty papular elevations could be seen or, even better, felt. There was generally considerable induration and thickening, and in parts where the cellular tissue was loose, œdema. It faded away towards the margin, and terminated sometimes with and sometimes without an abrupt edge, beyond which a few scattered papules could be seen.

(c) Another mode of commencement (14 cases, 8 males and 6 females) was with slightly raised blotches of congestion, having an abrupt margin and circular outline (like erythema nodosum, or *E. papulatum*, or R  thelu).

(d) In six of the cases the eruption commenced with one or more small flat papules, which, as each enlarged, presented an appearance resembling ringworm of the body, a circular spreading ring of congestion enclosing a depressed area covered by minute vesicles. The importance of these cases will be further alluded to.

About the second or third day in the moist type of case vesicles developed, though they could rarely be seen intact, for they were so easily broken. If they remained whole their contents generally became cloudy in the usual way. In one case, where the vesicles were of unusual size, I was fortunate enough to procure some of the contents of an unbroken one for bacteriological research. A cover-glass specimen, showing diplococci, is exhibited here to-night.

2. *Stage of Exudation or Desquamation* (lasting 3—8 weeks).—But whether vesicles were formed or not, and whether the rash began as papules or macul   they soon became confluent, and then presented the appearance of a crimson surface of thickened and indurated skin continually shedding its cuticle, in scales or flakes of various size, mingled with more or less dried exudation.

From the presence or absence of exudation I have grouped my cases into two varieties. In one, the “moist” type, the papules were followed by vesicles and copious exudation; while in the other, the “dry” type, there was no exudative stage, or, if so, it was very transient, the epidermis being thrown off without exudation, and the skin remaining dry throughout the whole course of the disease. About two-thirds of the cases were classed as “moist,” or eczematous. There were, however, very few pronounced cases which did not at some period of their history exhibit slight moisture either in the flexures of the joints or folds of the skin elsewhere.

The skin remained in this crimson inflamed condition for several days or several weeks, continually shedding its epidermis, not once or twice only but many times. The size of these flakes varied from impalpable powder to the entire cast of a hand.

3. *Stage of Subsidence*.—By degrees the inflammation subsided, leaving the skin thickened and indurated, but with a polished brown appearance. In many cases the skin looked raw or parch-

ment-like, smooth and shiny; sometimes with cracks here and there, not unlike ichthyosis vera.

In several of the cases the skin assumed a purpuric condition beneath the other elements of the eruption. This occurred for the most part in the later stage of the disease and amongst the aged.

LOCALITY.

The following Table A shows the relative frequency with which the various parts of the body were first attacked:—

TABLE A.—*Showing the Frequency with which various Parts of the Body were first affected.*

	Male. cases.	Female cases.	Total cases.	Percentage.
Arms and forearms	25	12	37	22·6
Face and scalp	15	20	35	21·4
Feet and legs (below knee) ..	11	13	24	14·7
Hands	15	7	22	13·4
Back	8	5	13	7·9
Neck	3	9	12	7·3
Chest or abdomen	7	5	12	7·3
Ears	3	2	5	3·0
Thighs and groins	2	2	4	2·4

There was a marked tendency to generalisation, but, inasmuch as all parts of the body were never simultaneously attacked, it follows that different places often presented different stages of the eruption. The rash seemed prone to start and predominate in the folds of the skin, such as the flexures of joints, beneath the mammæ, behind the ears, &c.

In exactly half of the cases the entire body was covered with the eruption sooner or later; but in some the disease was limited to patches. In such cases the extreme thickening of the affected skin was more obvious by comparison with the healthy intervening tracts. In a few of the younger cases the rash was extremely trivial.

Mode of Spread.—I was much concerned to discover whether the march of the skin affection was one of spread from contiguous parts, or whether it ever appeared simultaneously in two distant and wholly unconnected places. In this way I hoped to decide the

question whether the disease was due to a purely local influence creeping along the skin, and producing secondary general effects, or, on the other hand, to some general poison in the blood, or perhaps to some general nervous influence which, selecting the tegmentary tissue for its evil effect, acted on one or more distant parts of the skin at the same moment. Certainly, in the majority of cases, the spread took place from neighbouring parts, or such as might have been brought into contact by the movements of the patient or the bedclothes. Not a single clear illustration of the opposite method came under my notice which would stand the test of critical investigation.

The general rule was for the eruption to start from one or more points, and spread thence during the succeeding days; but in a few cases the rash burst out suddenly, or in the course of a few hours, over a large area, as in case M., 89; and this sudden involvement of large tracts is a point worth bearing in mind in connection with the question of pathology.

Subjective Symptoms.—These consisted of severe burning, itching, and irritation. After the loss of the epithelium the skin is very sore and tender to the touch. The peculiar sour odour exhaled from the severe cases (quite half of the patients) attracted everyone's notice. Sleeplessness was common in the earlier stages, but this was accounted for in most by the intense irritation of the skin, which was always worse at night.

The *other epidermal structures*—hair and nails—shared in the disease in its later stages. In many cases, nearly all in which the eruption had been severe and extensive, all the hair of the body was shed; and the nails often came away, or indicated by a rough transverse ridge the date of the disease.

CONSTITUTIONAL SYMPTOMS

Of some kind were present in nearly every case, though they were very slight in some. They seemed, as a rule, to bear some general relation to the extent and severity of the skin lesion, but by no means always. Of these, two symptoms, *anorexia* and *prostration* were by far the most constant. The weakness, or a feeling of exhaustion, in a few cases, preceded the rash by a few days, and in nearly all it lasted for a considerable time afterwards. In the severer cases the prostration was very pronounced, and tended

more than anything else to bring about a fatal issue. In certain cases (M., 11, 23, 38, &c.) the rash was of moderate dimensions, but the asthenia was very great, and lasted for weeks after the other had gone. Many of the patients suffered severely from thirst; and in all cases one or more of the three symptoms—*anorexia, asthenia, or thirst*—was always present.

The *temperature*, during the earlier period of the eruption, was generally normal, or in many of the older patients sub-normal. In the later stages, when the skin became extensively or severely inflamed, it went up to 99° or sometimes 100° in the evening, and generally down again to normal in the morning.

A higher temperature than this usually indicated the formation of boils or some other local inflammation. The most typical chart was a long course of intermitting *apyrexia*, *i.e.*, a temperature of about 97° or 98° in the morning, and about normal in the evening; with here and there a rise to 100° or 101° , lasting a day or two.

COURSE AND MODE OF TERMINATION.

The disease began and ended gradually, so that it was sometimes a little difficult to fix an exact date for either. This was more true of the ending than of the commencement. Very often the eruption was preceded for a few days by a sensation of irritation or tingling of the skin, unaccompanied by any visible signs. Sometimes it was preceded for a few days by a feeling of malaise and loss of appetite. (Case M., 27 and 38, &c.).

Very frequently a slight erythema or a few papules would appear, and, either stay in that condition for a week or so, or fade away, as though the attack would abort; but then taking on fresh vigour, would rapidly declare its serious nature. The termination in all cases was by lysis. Taking the end to occur when the primary or principal attack was over, the *average duration* amongst the males was 7·24 weeks, females 7·32 weeks, and in all cases 7·27 weeks.

Recrudescence or relapse was a common feature in the epidemic. It occurred in 38 of the patients, 24 had one relapse, 4 patients had 2 relapses, and 10 had 3 or more. One patient (M., 22) had 9 relapses, and one (M., 64) had 10. It is interesting to note that both of these patients continued to relapse until the termination of the epidemic, suggesting the idea that the termination of the disease in them depended on a disappearance of the

poison from the air around, and not an exhaustion of the pabulum within them.

Mode of Termination.—Among the constitutional symptoms were two, referable to the *nervous system*, which are worthy of special mention, as occurring only in the very worst cases; viz., twitching of the muscles (*subsultus tendinum*), and shallow or sighing respiration without other signs of pulmonary mischief. These symptoms, either alone or together, were of the gravest import.

All the eighteen patients who died of the disease, died in much the same manner. The weakness became profound, and was succeeded by torpor; the patient, however, generally retaining consciousness till within twenty-four or forty-eight hours of death. But, gradually, the drowsiness deepened into coma, which, without the supervention of convulsions, terminated the scene. The chief feature of the mental condition, towards the end of life, was torpor, though some cases were attended with nocturnal delirium towards the end. The patient could be roused to answer questions, but dropped off to sleep immediately. The symptoms here mentioned were very suggestive of the retention of an auto-intoxicating poison within the body, which acted on the nervous system.

COMPLICATIONS AND SEQUELÆ.

As the skin lesion subsided several of the patients (twelve or more cases) were troubled with *boils* or *carbuncles*, sometimes blind, but more generally, suppurating. One case (M., 85) had as many as nine carbuncles on different parts of his body at one time, and another (F., 75) had five boils on one arm.

As the flakes cleared off, the new skin was unduly tender and congested, which was only to be expected; but in several instances this was succeeded by a brownish or chocolate *pigmentation*; a condition well illustrated in one of the cases shown to-night. The pigmentation is very evenly distributed over the whole body; there is none on the mucous surfaces. The *falling off of the hair*, and the shedding of the nails already mentioned, continued for a considerable time (some months) after all signs of the eruption had gone.

The *conjunctivæ*, which were inflamed in all the severe cases, and those where the face was involved, discharged a highly-irritating sero-purulent fluid, and a similar acrid fluid came from the nostrils in a few cases. But the inflammation did not stop

here, for the iris was often involved; and many of the patients suffered from *irido-cyclitis* with severe photophobia. Several were troubled, for some weeks after all rash had disappeared, with recurrent attacks of conjunctivitis or iritis.

SYMPTOMS REFERABLE TO OTHER ORGANS.

Alimentary Tract.—After the eruption and the prostration, the total loss of appetite was undoubtedly the most pronounced feature in nearly all cases. It is noted as existing in a very marked degree in 51 out of 122 cases, or 41·8 per cent. The positive loathing of food, which often existed concurrently with the rash, was only equalled by the ravenous appetite which followed with convalescence.

The tongue at first was covered with a thick white fur; but very soon this coat came off, and generally left a red-raw surface during the remaining weeks of the disease, of the soreness of which the patient bitterly complained. In severe cases it became dry, the saliva thick and viscid, and sordes collected round the teeth. The appearance of the tongue was quite analogous to the skin changes, and the suggestion was irresistible that, in some cases at least, the epithelium of the alimentary canal shared in the lesion of the skin—an idea which was strengthened not only by the vomiting and diarrhoea during life, but by the appearance of the stomach and intestines in at least one fatal case (M., 1). Here, the mucous tract was covered by a whitish coat of thickened epithelium, that could easily be scraped off.

Diarrhoea occurred at some time in the course of the disease in 10 cases, and *vomiting* in 8, and 11 had both vomiting and diarrhoea, out of 80 cases in which sufficiently detailed notes were made on this point. Diarrhoea or vomiting occurred chiefly in connection with those cases that appeared during the later months of the epidemic. It is interesting to note the incidence of these symptoms on different sexes. Out of 56 males, 7 had vomiting, 7 had diarrhoea, and 7 had both. Out of 24 females, only 1 had vomiting, 3 had diarrhoea, and 4 had both, showing that these symptoms are less liable to occur in the female. These symptoms did not occur at any constant period in the course of the disease. In some one or other of these symptoms came on several days before the appearance of the eruption; in others they happened when the skin lesion was at its height, while in many they occurred

during convalescence, when the skin was clearing, or even when it had quite recovered. It was the uncontrollable vomiting and diarrhoea which brought about a fatal termination in case M., 25. In this instance there was also incontinence of urine and fæces, an evidence, probably, of the extreme prostration to which the patient was reduced.

The urine was examined from time to time in seventy-two cases and albumen was found in thirty-six, or exactly half. The albuminuria only appeared when a considerable area of skin was involved, and only when the eruption was at its height, or during the later stages of the disease. This is not a surprising circumstance when one considers the large amount of additional work thrown on the kidneys. The albuminuria was probably the result of congestion of these organs; at any rate, in the event of recovery, no signs of organic disease remained, and in the fatal cases deep congestion was discovered. In many, but not all, of those who had previous signs of organic renal disease, the eruption proved fatal.

Lungs.—In most of the fatal cases which were examined after death, the lungs showed intense *hypostatic congestion*; but inasmuch as the immediate cause of death was either asthenia, in most cases, or uræmic coma, this condition of the lungs is rather to be regarded as a part of the latter than a complication of the disease. Some of the patients were attacked with *pleurisy* during the course of the disease. *Pneumonia* occurred in the course of case M., 1, and he rallied from it, but did not recover ultimately. It also occurred in some others.

The *heart* gave evidence of sharing in the general debility, and the pulse was usually feeble and of low tension, often intermittent.

The *purpuric basis of the eruption*, to which reference has already been made, may be regarded as a vascular complication of the disease. Sometimes, as in case of M., 78, it showed itself after the skin had recovered. In that case the entire body and limbs became covered with thickly set spots of extravasated blood, varying in size from a split pea to a large bean.

VARIETIES.

As regards the clinical varieties of the affection, beyond the two types, founded on the “moist” or “dry” character of the eruption,

already referred to, no other distinct varieties need be recognised. The division, however, into these two classes is a legitimate one, for there are other features which separate these groups besides the character of the rash. Of the total number attacked up to date (November 1st, 1891) 100 belong to the moist type, 45 to the dry type, and 18 were of a mixed type, different parts of the body presenting different varieties of the eruption. The two varieties may be conveniently called *Dermatitis Humida*, and *Dermatitis Sicca*.

It would seem that the variety of eruption was determined partly by the idiosyncrasy of the patients in possessing what might be called a dry skin, or a moist and easily perspiring one; for the former mostly resulted in dry or pityriasis cases, and the latter moist or eczematous ones. The dermatitis humida usually ran a more rapid course than the dermatitis sicca, which was nearly always prolonged and chronic; moreover, so far as I recollect, the weakness was a more marked feature of the dry variety than of the moist, possibly on account of the ages of the sufferers. The table showing the numbers and ages in the respective groups may need revision, but it shows the average age of the humid cases to be 62·6 years, and that of the dry to be 68 years, as compared with the 64·8 which is the average age of all the patients attacked.

Although it is inexpedient to make any other division than the one mentioned, still, as I have already said, the cases differed considerably from one another in detail, and especially in the extent and distribution of the rash, and in the severity of the constitutional symptoms. It is of great importance to bear this in mind in searching for cases of the disease, and especially when it is a question of an isolated case. A few of my cases had only a small amount of redness or scurfiness, localised, perhaps, to one side of the face, or, as in my own attack, to the ears, or to the hands, or the flexures of the joints. In these the constitutional symptoms were generally, but not always, of quite a trivial order, and it is most important to observe that these, if seen alone, apart from the epidemic, might (except, perhaps, for the exfoliation) have been taken for eczema.*

* As an instance, a case may be mentioned which I have since seen with Dr. Evans. A little girl of 8 had developed, three weeks before, a redness of the face and head, which soon became scaly. The rash spread on successive days to other

DIAGNOSIS.

Bearing in mind the variation amongst the cases, it is not surprising that some of them required most careful differentiation, although a well-marked type presented no great difficulty. The leading feature of all, *the pathognomonic symptom*, was the *desquamation or exfoliation of the epidermis*, and it is necessary to observe that this occurred sometimes, in some parts, without any previous rash.

A. *Erysipelas*.—Where the face was the only or the chief part involved, or, indeed, any part containing much loose cellular tissue, the tumefaction of the parts, sufficient, sometimes, to close the eyes, bore a strong superficial resemblance to erysipelas; but the gradual advent, exceedingly limited range of temperature, the often vesicular nature of the eruption, and the fact that the rash occurred elsewhere in a more typical manner, sufficed to distinguish these cases from erysipelas.

B. *Rötheln*.—We have seen that the rash in fourteen cases began with blotches, and in these cases, before the maculæ became confluent, it was exceedingly suggestive of German measles. But the persistence of the eruption, its taking on an eczematous or scaly character, and the absence of pyrexia, served in general terms to distinguish these cases from Rötheln.

C. *Pityriasis rubra*.—The fact that the disease was in all cases an exfoliative dermatitis, linked it with this class of malady; and all the cases belonging to the “dry type” had a striking superficial resemblance to the descriptions of pityriasis rubra of Willan and Wilson.*

parts of the body, so that at the end of a week all but the legs below the knees had been affected; lastly, these and the feet were involved. The epidermis generally was exfoliated in flakes and scales, which were shown to me, of various sizes, that of the hands and feet in pieces an inch or more. Large pieces were coming off the feet at the time I saw her. Nothing but scarlatina could have produced such a widespread exfoliation, and this was negatived by the slow march of the rash, the total absence of pyrexia, sore throat, characteristic tongue, or contagion to a large family of brothers and sisters. There was no history of anorexia, thirst, or any constitutional symptoms at all in this case, and, except for the desquamation, the little patient suffered no discomfort whatever. The eyes had been a trifle sore at one time. How widely different is such a picture from that of some of my elderly male patients! Yet it was evidently the same disease.

* ‘Diseases of the Skin,’ 5th edit., 1863, p. 111.

But, on the other hand, my cases were evidently contagious, or, at any rate, occurred in an epidemic form, whereas no mention is made of this in the descriptions by these authors.

2ndly. Dr. Liveing says, "the affection (pityriasis rubra) is very commonly met with in children and persons possessed of a delicate skin and fair complexion"; whereas the great majority of my cases occurred in adults and old people. There were very few amongst children, and these very slight (*vide* table, p. 119).

3rdly. There is the difficulty of reconciling the moist cases (which were in the majority) with the recognised type of pityriasis rubra. Liveing, Crocker, and others describe the skin in pityriasis rubra as not infiltrated or thickened, but in my worst cases the skin was very decidedly so, and in all there was distinct induration. Nevertheless, in many respects, these cases tally with Liveing's description.

"Pityriasis rubra," says Dr. Liveing (p. 98),* "is really a rare and peculiar form of eczema as it is now regarded by Erasmus Wilson, Neumann, and even Hebra himself, who was the first to describe the affection as a distinct disease."

"It differs from common eczema chiefly in the entire absence of moist exudation on the surface of the skin, and in the extraordinary exfoliation of cuticle. The first distinction I consider more apparent than real, for I have detected traces of dried exudation on the under surface in several cases." It may be here mentioned that I examined some of the flakes shed by those of my cases apparently quite free from any exudation, and by the aid of a lens, dry secretion was distinctly visible on the under surface.

D. *Eczema*.—Let us inquire in what respects my cases resemble and differ from eczema, more especially in its Acute General form. They are alike in that:—

1. Both diseases start as a papular, or papulo-erythematous rash.
2. Both diseases are prone to become vesicular, and to be attended by exudation. We have seen that altogether 118 cases out of 163 had a definite amount of exudation.
3. Both diseases have a marked tendency to select as starting places the flexures of joints, and folds of skin, such as those beneath the mammæ, or behind the ears; and here also in many of my cases the rash predominated.

* 'Diagnosis of Skin Disease,' 1878, p. 100.

4. Both diseases are apt to begin in one part of the body, and then as a fresh patch appears in another part, the first one either runs an independent course or fades away; and thus different parts of the body present different stages of the eruption.

When I first drew attention to this disease in July, 1891,* I myself fell into the error, and so did others† who saw the cases in an early stage, of supposing it to be a variety of eczema, hitherto undescribed, occurring in an epidemic form. But further scrutiny and the observation of many more cases, especially in a more advanced condition, have led me to doubt whether it is eczema in any form; and this view is now shared by many others‡ more competent than I to judge.

The differences between the two diseases are:—

1. The amount of dermal thickening and induration in nearly all of my cases was certainly far greater than is seen in eczema of even a severe kind (*vide* illustration).
2. The exfoliation of large flakes of epidermis, which so frequently occurred in both moist and dry cases, is certainly very unlike acute general eczema. That a proportion of these cases (about one-third) were apparently free from exudation from beginning to end is not a point on which too much stress should be placed, for, as we have seen, exudation could be discovered on the under surface of the scales.
3. The definite course of six to eight weeks, which most of my cases ran, is certainly a notable feature. They were, like eczema, liable to relapse, but the primary attack ran a definite course, which differs widely from the clinical history of eczema, either localised or general, so far as I am aware.
4. In cases of acute general eczema there is generally a certain amount of malaise, but nothing like the severe constitutional disturbance present in the majority of my cases and which was the immediate cause of death in 12·8 per

* 'Lancet,' August 1, 1891, p. 266.

† Correspondence in 'Lancet,' August 8, 1891.

‡ Discussion at Medical Society of London, 'Lancet,' December 5, 1891, p. 1279.

cent. Sometimes the constitutional disturbance was out of all proportion to the eruption, even to the extent of proving fatal, as in the remarkable case of M 38, where the rash was both trivial and transient.

5. Eczema attacks all ages, and especially the delicate skins of children, but this disease, as we shall see, is almost entirely confined to persons at or beyond the middle period of life (table, p. 119).
6. The occurrence of this disease in an epidemic form distinguishes it at once from any variety of eczema hitherto described.

Nevertheless, the resemblance of the skin lesion in some of my cases to Eczema, and in certain others to Pityriasis Rubra was so remarkable as to render it *highly probable that this disease, at least when occurring in a sporadic form, might be, and probably has been, classed as one or other of these diseases.*

It is possible that there are other diseases from which my cases have to be distinguished, or which they resemble, but, I have purposely abstained from reading up authorities, lest my mind should become confused or biassed in the observation of the cases and in the realisation of their true type.

PROGNOSIS.

In twenty-one out of my 163 cases, death was a direct consequence of the disease, giving a death-rate of 12·8 per cent. Seven others died, but the immediate cause of death was some other condition. Nevertheless, this is a higher mortality than in any other contagious malady now prevalent in the United Kingdom, excepting perhaps typhoid fever.

We have seen that more males were attacked (eighty-nine males and seventy-four females); the male cases were also more severe, and the mortality was much greater in the male sex (20·22 per cent. amongst the men, 4·05 per cent. amongst the women).

Age seems to have had an important influence both on the severity and the mortality of the affection. The youngest who died was 49, the next 59; and the average age amongst the twenty-one fatal cases was $71\frac{1}{4}$ years, as compared with 64·8, which was the average age of all attacked.

The degree of weakness and prostration was a very fair indica-

tion of the probable course of a case, both as to its duration and issue; and there were two signs which were, as already mentioned, of the gravest import, tremor or twitchings of the muscles, and laboured respiration without physical signs of lung mischief. Not one of the patients in whom I observed these symptoms recovered, although, in some cases, they seemed at the time to be doing well in other respects.

TREATMENT.

Many different kinds of treatment were employed, but when the disease had once become fully established, nothing beyond amelioration could be effected. As a local treatment, a lotion of creolin or carbolic acid seemed to be efficacious in some cases, and in two or three instances, where it was used quite in the initial stage, the disease was to all appearances cut short. Even at a later stage, this lotion or a creolin ointment made up with lanolin, seemed to have a remarkably soothing effect on the irritable inflamed skin, and some cases improved considerably under it. But, on the other hand, in certain cases it seemed to do harm. The irritation from which so many suffered was often relieved by warm soda baths, or bland emollient ointments and lotions, such as vaseline, zinc ointment, calimine lotion, lead lotion, &c. No internal medication was of much use, but stimulants were distinctly indicated, and in more than one instance large quantities of whiskey appeared to avert a fatal issue.

ETIOLOGY.

A. Predisposing Causes.—1. *Age.* Most of the sufferers were in advanced life; indeed there were none but quite trivial cases in children and young adults. The average age of all males attacked was 63·67, and of females 66·17, and of both sexes 64·8. But not only were all the pronounced cases in persons past the middle period of life, but instances might be mentioned, where, of several patients equally exposed to contagion, the younger ones escaped, and the elders contracted the disease. More than one example is within my recollection, where the disease in its spread along the side of a ward would skip over a bed containing a younger person. Nevertheless, it may be objected that there is a larger proportion of elderly people in the infirmary; and therefore it was

TABLE B.

Showing the Proportion between the Number of the Cases at a Certain Age, and the Number of Persons in the New Infirmary at that Age.

Age	1-9.		10-19.		20-29.		30-39.		40-49.		50-59.		60-69.		70-79.		80-89.		Totals at all ages.	
			M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Number of patients under treatment at that age }			27	29	8	16	24	33	28	18	29	29	26	17	43	30	37	42	5	14	227	228
			1	0	0	1	2	2	2	1	8	2	9	2	16	12	18	9	1	4	57	33
Proportion between cases of the disease to patients under treatment at that age, per cent. ... }			3.7	0	0	6.25	8.33	6.06	7.14	5.55	27.58	6.89	34.61	11.76	37.20	40.00	48.64	21.42	20.00	28.57	25.11	14.47
Proportion attacked in both sexes combined {			1 out of 56 = 1.78 p. c.		1-24 = 4.16 p. c.		4-57 = 7.01 p. c.		3-46 = 6.52 p. c.		10-58 = 17.24 p. c.		11-43 = 25.58 p. c.		28-73 = 38.35 p. c.		27-79 = 34.17 p. c.		5-19 = 26.31 p. c.		90 out of 455 = 19.78	

necessary to know the number of inmates in each decade of life, who were equally exposed to the contagion, so as to ascertain the relative proportion of each attacked.

The table (p. 119) prepared by my colleague, Dr. E. V. Hugo, gives the information required.

From this it will be seen that while only 1, 4, 7, and 6 per cent. were attacked among the earlier decades of life, 17, 25, 38, 34, and 26 per cent. suffered in the decades from 40 upwards. Hence, one may fairly conclude that advanced life was an important predisposing cause of the disease, and that the predisposition gradually increased as age advanced.

2. *Sex*.—The above table also shows that out of a total of 227 males under treatment in the infirmary 25·1 per cent. were attacked, but out of the 228 females only 14·4 per cent. were attacked. The figures of the workhouse sick wards do not show quite such a contrast, for of the males 22·14, and the females 16·94 per cent., were attacked. If both places be taken together, it shows that 23·67 per cent. of the total males, and 15·74 per cent. of the total females were attacked; showing that among my cases the male sex exhibited a marked predisposition to the disease.

3. As to the *occupation* of the patients, it seemed to have no particular influence apart from other causes.

4. *Previous Ill-health and "Hospitalism."*—Among the sick inmates of the infirmary 19·7 per cent. were attacked, but among the healthy staff only 2 (myself and a housemaid) or 3 per cent. Again, out of 391 sick inmates of the workhouse sick wards 18·6 per cent. suffered from the disease; whereas, out of 169 aged and infirm but healthy inmates who belonged to precisely the same class of life, and were exposed to precisely the same environments, only 11 were attacked, or 6·5 per cent. And of these latter 7 (2 males and 5 females) were "helpers" in the sick wards, and constantly tending on patients already suffering from the disease. From these facts it would seem that sickness and "hospitalism" are almost as important factors in the causation as advancing years.

5. Careful examination of a tabulated statement of *the disease for which the various patients were under treatment* at the time when they were attacked, did not lead to the suggestion that any particular disease was more prone to be complicated by the eruption than another, with the possible exception of ulcer of leg. A few

of the cases exhibited evidences of a gouty diathesis, either in their own or family history, but not a sufficient number to show any connection.

Some of the epidemic cases had had *influenza* during the twelve months preceding the outbreak, but, so far as I have been able to discover, this produced no marked predisposing influence. It has been suggested that the epidemic eruption was one of the various manifestations of influenza, but I have been unable to trace any evidence in support of such a supposition.*

B. *Exciting Causes*.—1. Turning next to the possible exciting causes of the epidemic, the first to be examined is *food*. A careful examination of a table showing the articles of dietary partaken of about the time when they were first attacked, prepared for me by Miss Annette Benson, M.B., the Clinical Assistant, entirely fails to connect any particular article or articles with the epidemic, and the mere fact that precisely similar cases occurred at the Marylebone Infirmary, St. Mary's Hospital, and elsewhere, where not only was the dietary scale different, but the articles were procured from different sources, is alone sufficient to preclude the dietary from any participation in the etiology. It may not be out of place to remark that the dietary at the Paddington Infirmary is exceedingly liberal, and prepared with greater care than is usual. The food, both in quantity and quality, is superior to any hospital or infirmary with which I am acquainted.

2. *The soap* was suspected by some, and enquiry made. But the same soap was in use at the time of the outbreak that had been used for a long time previously without harm.

* *Premonitory Stage*.—I owe a great deal to the interest which the "sisters" and nurses of the various wards have taken in assisting me in this enquiry, and the observation of one of them (the sister of Ward V) ought, I think, to be recorded, namely, that five of her patients complained for a week or so before the appearance of the rash of symptoms exactly resembling influenza, but without any elevation of temperature, namely, headache, pains in the limbs and across the loins, loss of appetite, and a feeling of sickness without actual vomiting. In another ward (Ward VIII), which contained a good many elderly bed-ridden cases, the "sister" noticed a very marked falling off of the appetite for several days before there were any signs or symptoms referable to the skin. Another "sister" (Ward VII) declares that in addition to the feeling of irritation on the skin which so many of her patients described before the actual rash appeared, they also complained of feeling very tired, of pains across the loins and down the limbs, without any elevation of temperature for five or six days' before the eruption was visible.

3. *Scabies* was another suggestion as the cause in the earlier days of the epidemic. But no burrows or other signs were ever found, though careful examination was made.

4. *The water supply* as a source of evil, either internally or externally, was also excluded.

Moreover, if any local irritant like soap, scabies, or water, were the exciting cause, how is it:—

a. That the delicate skins of children and infants escaped in so marked a manner, while the proneness to the disease increased *pari passu* with advancing years?

b. That the parts first and chiefly attacked by no means always corresponded with the parts most exposed to local influences?

c. That the Marylebone Infirmary obtaining its food, water, and soap from different sources should, at the same time, be subjected to a very similar outbreak as the one we are considering.

The question therefore remains: Is the disease due to some obscure *epidemic influence* such as climate, atmosphere, season, or contagion?

5. *Climatic or Atmospheric Causes*.—It certainly is a remarkable circumstance that the two neighbouring Metropolitan infirmaries, which are the newest, and which are therefore, if I may say so, renowned for their sanitary perfection, should be thus subjected to the ravages of the disease. A few cases occurred, I believe, at Lambeth, but nothing like the outbreak at either of the other two. The other twenty-two Metropolitan infirmaries seem to have been exempt, or almost so.

6. *Season*.—A superficial examination of the occurrence is somewhat suggestive that an obscure seasonal influence, though certainly not the proximate and only cause, might have taken some part in the etiology; for at Paddington and Marylebone the outbreak was strictly limited to the summer and autumn months. Further, Mr. Lunn informs me that cases of a similar disease occurred at Marylebone last autumn (1890), only of a milder type and fewer in number. The following table shows the numbers which were attacked in the various months; from which it will be seen that the greatest number occurred in July and August:—

TABLE C.

			May.	June.	July.	Aug.	Sept.	Oct.
Males	2	11	37	29	6	4
Females	0	9	22	27	9	7
Totals	2	20	59	56	15	13

7. *Contagion*.—There are many features in which this resembles a specific contagious disease; indeed, except for the absence of pyrexia, it might almost be grouped with this class of disorder. The absence of fever is a point of considerable importance, and will merit further investigation. Nevertheless, there are certain features in the clinical history of the epidemic, and of the disease, which prove it to be contagious, and due to the presence of a specific living organism:—

(i.) The large number of cases attacked at one time, nearly 20 per cent. of the patients.

(ii.) The more or less definite course of seven or eight weeks through which the primary attack runs in well-marked cases of the disease.

(iii.) Although, as we have seen, the cases differed widely in detail, the marked general resemblance between them all was a fact which struck the most casual observer, and the inference therefore is that the cause, whatever it be, is specific.

(iv.) The symptoms of constitutional disturbance which attended the skin lesion.

(v.) The three features, together with the existence of a cutaneous eruption, complete the resemblance to an eruptive fever.

(vi.) The serpiginous rings with which some of the cases started bore a strong resemblance to *tinea circinata*, a malady known to be due to a specific contagium.

(vii.) The marked effect of germicides in modifying the skin lesion when applied at an early stage.*

(viii.) The wave-like manner in which the outbreak rose and fell, strictly limited to the summer and autumn months of 1891.

* A similar fact of much importance was mentioned by Mr. J. R. Lunn in the discussion which followed. If collodion were applied to a patch of the eruption in an early stage, shutting up the contagion as it were, it checked the progress of the disease.

(ix.) Clear instances of direct contagion are always difficult to establish, but it is worthy of note that 6 of the 11 persons who, out of 193 healthy aged inmates of the Workhouse proper, contracted the disease, were acting as helpers or "pauper-nurses" tending on, and in direct contact with, patients already suffering from the complaint. The facts that I and my little dog (who is in the habit of accompanying me on my daily round), contracted mild but undoubted attacks of the disease are particularly interesting in this connection.

(x.) And lastly, the known existence of a specific contagion in the shape of a diplococcus.

Nevertheless, the disease is not malignantly contagious, for it does not, as we have seen, attack the sick and healthy indiscriminately. Certain predisposing conditions would seem to be absolutely essential to the propagation of the disease, chief among which are age and previous sickness. Whether there exists a period of incubation, and whether one attack confers immunity, are questions which cannot be answered at present, but there would seem to be a prodromal period in some cases (*vide* foot-note, p. 121).

There are certain features which seem to be highly suggestive, though not more than suggestive, that the specific contagium, whatever it be, attacked the unbroken skin and, setting up inflammation there, was introduced into the system by that means.

1. The ringed serpiginous character of the eruption at the outset which was definitely observed in several cases.

2. The marked effect of local germicides in controlling the disease, if externally applied in the early stage of many of the cases, and of absolutely cutting it short in at least one case.

3. The eruption showed a tendency to start on exposed parts, such as the face, forearms, and hands, and to spread thence.

4. The proneness of the eruption to assume an eczematous character, eczema being the form of skin disease which most often arises from local irritation (*e.g.*, "baker's itch," &c.).

There still remain many important questions to consider; but these must be deferred to a future occasion.

In conclusion, I wish to acknowledge the valuable assistance I have received from my colleague, Dr. E. V. Hugo, in preparing several of the tables and in investigating the clinical phenomena of the outbreak; from my assistant, Mr. Clark, in helping me with the series of photographs; and, lastly, from the nursing staff in

procuring precise and detailed information concerning the various patients.

And now I would venture to seek aid in the explanation of the various facts set forth in the foregoing pages.

1. What is this strange disease? Is it a totally new condition which has never visited this country before? Or is it an old disease (eczema, for example) modified by the circumstances of time, place, and individual? Or, again, is it a compound of two conditions (*e.g.*, erysipelas and eczema) usually met with separately?

2. Is the disease of the nature of a constitutional eruptive fever, or is it a local inflammatory disorder, having secondary general effects?

3. What are the lessons to be learnt from this strange outbreak, and what are the steps to be taken to prevent its recurrence?

4. In what other places have similar cases occurred; what are the local causes in action, and what are the real limits of the epidemic?*

It was with the object of procuring valuable criticisms on these and other points that I have so long occupied the time of the Society, and it only remains for me to thank you for the patient and courteous hearing afforded me.

Mr. J. R. LUNN: From May, 1891, until the 25th of the following October there were 193 cases of epidemic eczema in the St. Marylebone Infirmary, Notting Hill, 105 of which were males, and 88 females. The deaths of 23 were certainly accelerated by the rash. The outbreak was most irregular in its attacks among the patients. Officers, nurses, scrubbers, and porters were attacked, but they did not suffer from the asthenic type. The question of diet, atmospheric changes, drainage, soap, local irritations, and cleanliness were thoroughly gone into. The rash appeared in those wards which had been lately cleaned and white-washed, as well as in those which had not been done. Isolation was tried at the onset of the outbreak; however the disease still spread through the wards.

* Since writing the above, several other instances of the disease have come to my notice, besides the cases under my own care, those at Marylebone in 1890 and 1891 (mentioned by Mr. Lunn during the discussion, 'Lancet,' 1891, vol. ii, p. 1279), and at St. Mary's Hospital (mentioned by Dr. Lees, in a letter to the 'Lancet,' 1891, vol. ii, p. 461), viz., twenty-eight cases at Hanwell Asylum (mentioned by Dr. Richards during the discussion), several cases at Lambeth Infirmary in 1889 and 1891, and sporadic cases at the Lock Hospital, St. Saviour's Infirmary, Shoreditch Infirmary, a patient near Addison Road, under the care of Mr. Stephen Paget, and one near Ladbroke Grove, under the care of Dr. Turnbull.

In some of the cases vomiting and diarrhoea appeared at the beginning, but most of them complained of severe itching and a feeling of tightness over that part of the skin where the rash appeared. It was first noticeable on the neck and forearms, then on the legs, and, in some cases, over the scrotum, followed by a dry dermatitis. The character of the rash at the beginning was papular, and, after a few days, vesicular, some of which burst, and were followed by weeping eczema, while others appeared to dry up, and were followed by desquamation in large flakes. The rash in a few cases in the early state somewhat resembled German measles, pityriasis rubra, and lichen simplex. Several of the cases had a rise of temperature, and had several successive attacks of the rash, followed by severe complications, such as meningitis, alopecia, gangrene of toes, severe form of conjunctivitis, with iritis, producing photophobia, thrombosis, pigmentation, diarrhoea, and vomiting, and a falling off of nails. Nothing unusual was found at any of the autopsies.

A variety of internal and local treatments was tried.

MR. J. PEEKE RICHARDS said that the institution with which he was connected, viz., the London County Asylum, at Hanwell, was in the same district as, although a little further west than, the Paddington Infirmary. He was in charge of the female department, and the average daily number of patients under his care was about 1,140. Early in July of the present year, several cases of a peculiar rash manifested itself among the patients; but, prior to this date, there may have been several cases of a similar but milder character, that had been overlooked. The recorded instances were 38, or 3 per cent. of the average numbers resident. The ages, arranged in decades, were as follows:—5 were between 30 and 40, 6 between 40 and 50, 12 between 50 and 60, 8 between 70 and 80. The disease occurred with the greatest frequency in the months of September and October. At the close of the latter month no fresh cases were noted, and the disease seemed to die out. The eruption, as a rule, commenced on the face and neck; it was at first papular in character, and then became vesicular. At this stage the whole face was reddened and swollen, and often had an erysipelatous appearance; afterwards the skin took on an eczematous character, and there was an exudation of thin serous discharge, issuing from small, cracked fissures. In several instances the back and loins were much affected. The skin generally became thickened and swollen, and often a scaly appearance of certain parts of the body was noticeable, much resembling pityriasis. There were no boils, but a little pigmentation of the skin occurred, but not to such a marked degree as in Dr. Savile's cases. In only one instance were the nails shed. As to treatment, those who were left alone recovered, and those who were treated with simple remedies, such as zinc ointment and lead lotion, did well. Nothing wrong could be traced with the food supply, and the water drunk by the patients came from a different source to that supplied to the inmates of the Paddington Infirmary: the Asylum deriving its water supply from a deep well which supplied the Institution only. As the skin disease began to die out, an epidemic of diarrhoea and sickness set in amongst the patients generally without any assignable cause. The disease did not appear to be contagious, and the cases were scattered about all over the building, not many instances occurring in one place, perhaps only two or three at the most appearing in a ward containing sixty patients. As to the etiology of the disease, Mr. Richards was inclined to believe that it was due to some influence of the same character as that which produces influenza, the germ acting upon that part of the nervous centre which controls and modifies integumentary changes.

Dr. C. M. CAMPBELL said that the chief interest of this series of cases lay in their etiology and pathology, and he regretted that no *post-mortem* information had been furnished, especially in regard to any microscopic examination of the spinal cord. The clinical phenomena bore a very close resemblance to those exhibited by a case of chronic exfoliative dermatitis, which had been under his observation for four years. Beginning when the patient was a boy of ten, it lasted with occasional remissions for twenty years, when he died. Death was preceded by atrophy of the muscles, acute general arthritis and motor paralysis. At the autopsy the spinal cord was found to be affected by extensive myelitis, as well as exhibiting heterotopia of the grey matter. A paper by Dr. Aldren Turner and himself, fully describing the abnormal phenomena, both central and peripheral, would be found in the 'Transactions of the Pathological Society' for 1891. The marked preponderance of cases occurring in the aged, in Dr. Savile's series favoured the hypothesis of a neurotic origin, as did also the occurrence of gangrene of the toes. Preceding languor and malaise, without pyrexia, did not resemble the prodromal symptoms of any of the contagious exanthemata; and did not support the view of a surface focus of infection gradually spreading through the organism. Argument in favour of the disease being essentially bacterial, based upon the presence of micrococci in the serum of vesicles was inconclusive, in the absence of inoculation experiments with pure cultivations of the cocci. Cocci were commonly found on the skin and in its secretions, but were not necessarily pathogenic. Certain species might perhaps change their qualities and become so, as a result of changes in the quality of the soil in which they grew. Still many of the facts suggested a bacterial contagion and it was possible that both atrophic nerve disturbance and a particular micrococcus were equally necessary and jointly responsible for the establishment of this disease.

Mr. JAMES STARTIN said, through the kindness of Dr. Savile, after he had carefully examined the cases of so-called epidemic eczema, in the Paddington Infirmary, and some that had come under his own observation at the London Skin Hospital, he had come to the conclusion the eruption in all respects partook of the nature of eczema, pityriasis rubra, or exfoliative dermatitis, and erysepilas, in varying degrees of similitudes. He dwelt, however, on the importance of recognising the differential diagnosis in these eruptions. Suggesting that most of the cases had prominent characteristic symptoms of *both eczema and superficial erysipelas*, (Crocker), suggesting a bacterial rather than a neurotic origin, and proposed to name the disease "*superficial erysipelalous eczema*." He considered the causes to be certainly external and septic rather than internal, and that the eruption was of a serious and fatal character, more prevalent amongst old people than young, and more common amongst men than women. He had found a soothing antiseptic treatment the most beneficial in the cases under his care.

Dr. STEPHEN MACKENZIE said that as regarded his own experience the outbreak was quite unique. He pointed out that the initial eruption in the majority of cases was papular; in the next series it was macular or erythematous, gradually spreading over the whole body. The features were those of an universal erythema with copious desquamation. He pointed out that the term "exfoliative dermatitis" was descriptive, and therefore preferable as involving no theory as to the mode of production. Beginning as a papular or erythematous eruption, it infected the whole skin, and ultimately the dermal appendages. He recalled that this con-

dition of exfoliative dermatitis was well known to them, though a comparatively rare disease. He had published in the 'Journal of Dermatology' twenty-one such cases, and he had pointed out how these cases might be caused, the mode of origin being different in different cases. Some began as eczema, some as psoriasis, but there were primary cases of general exfoliative dermatitis which in others was only a later condition. The interesting feature in connection with his own cases was that they occurred sporadically, the sporadic cases differing in no essential particular from the disease in its epidemic form. These sporadic cases had been admitted into general wards, and he had never seen any spread of the disease. He urged, therefore, that there must have been some special conditions in the environment to account for the outbreak. He, himself, had always regarded eczema as a purely local disease. He would be prepared to accept any bacterial theory if sufficient grounds could be shown. He said that the sporadic cases nearly all occurred after middle age, the liability increasing with the advance of years. He was unable to say whether it was a new disease. He raised the question as to whether the clothing might not have had some share in its dissemination; he understood that in such institutions, as in the early Christian communities, these things were in common, and he would like to know what precautions were taken for cleansing and disinfecting the clothes.

Dr. CROCKER said he had examined a considerable number of the cases, and it seemed to him that there was nothing characteristic about the eruption itself. He pointed out that eczema might develop from a single point; he had seen an ordinary blister prove the starting point of a general eczema. He agreed with Dr. Mackenzie that pityriasis rubra might start from any kind of dermatitis, and whenever the cutaneous envelope was affected extensively, the patient was always in considerable danger. The large number of fatal results occurring in old people with broken-down constitutions was therefore not surprising. He raised the question whether it was a general disease like the exanthemata, of which the eruption was only a part, or a local disease, the general symptoms being merely incidental. There were facts in support of both views. He had noticed that there was enlargement of the glands in a considerable proportion of cases. Of thirteen cases he had examined, nine had enlargement of the occipital glands, and in some of the others the glands behind the sterno-mastoid and in the sub-occipital region were enlarged; the enlargement, moreover, was not in proportion to the dermatitis. This was also observed as a constant feature in *rötheln*. He pointed out that, most of the patients being in bed, it would be easier to infect other parts of the body than if they were up and about. The cases generally began with one patch, subsequently becoming general, which was not unusual in pityriasis rosea. He inclined to the view that the disease began from without and became general. That naturally led one to imagine that it must be due to some organism or its product, probably the latter. Against its being a general disease was the fact of its being endemic rather than epidemic. How it originated it was impossible to say, but, having started in these places, one could easily conceive of the clothing or air becoming impregnated with the *materies morbi*.

Dr. DOWNES, Poor-Law Inspector, had availed himself of his connection with provincial workhouses to inquire of the medical officers at 120 of these institutions, none of them containing less than 100 beds for the sick, and he was indebted to these gentlemen for their kindness in replying. The returns which he had received related to some 25,000 beds

occupied on an average by 20,000 patients, so that a very large number of sick and infirm persons must have passed under the observation of the medical officers during the current year—the period specified in the inquiry. It appeared that with perhaps one exception—a workhouse in the west, with about 150 sick, where there had been “an unusual number of cases of eczema and a few cases of pityriasis rubra”—no approach to the condition of things observed at Paddington and Marylebone had been noticed, although the sources of information embraced the whole of England and Wales outside the metropolis. This was confirmed by his personal observation during an extensive series of workhouse inspections in all parts of the country. Indeed he had been impressed by the comparative rarity of exfoliative dermatitis of any kind. He had lately seen a case in which myelitis had apparently followed on eczema. The patient was an agricultural labourer, aged 24, previously healthy, who had about 6 months before been attacked with eczema which gradually became general. Some two months later, while returning from work, he fell down in some sort of fit and was taken to the county infirmary, whence he was eventually removed to the workhouse. Paralysis, more or less complete, of all his limbs had supervened. Dr. Downes particularly noticed the peculiar odour in this case resembling that described by Dr. Savill. The case in question, however, was apparently quite sporadic. He pointed out, in conclusion, that the inmates of provincial workhouses were chiefly old people and would, therefore, according to the statistics, be predisposed to this affection.

Dr. SAVILL, in reply, said he wished to correct the impression that most of the cases had started on the face. Only 22 per cent. had begun here, 23 per cent. on the arms and forearms, 8 per cent. had started on the back, 14 per cent. on the legs. His paper had been confined to the clinical aspect of the complaint. He had not at present had time to fully investigate the anatomy and pathology of the disease. He was unable to trace any distinct connection between the affection and influenza in his cases; nor did he see any reason to attribute the cause to a morbid influence of the nervous system. Beyond all doubt the malady was contagious, and probably bacterial in origin. The author and his little dog had both contracted attacks of the malady. He had devoted much time to the bacteriology, and had found a specific diplococcus constantly present in the serum and exudation of the patients. A specimen of this, derived from an unopened vesicle, was shown under a microscope on the table. The microbe resembled *Staphylococcus albus* in some respects, but differed from it in giving a thinner and whiter culture, which did not liquefy gelatin, at any rate for a considerable time. The cocci *always* occurred in pairs. He had once succeeded in producing the disease by inoculation of a culture into a rabbit; but too much importance should not be attached to a single instance.

December 7th, 1891.

ON CERTAIN CARDIAC SYMPTOMS OBSERVED IN CASES OF GASTRIC ULCER.

By WILLIAM M. ORD, M.D., F.R.C.P.

SOME two years ago, at a meeting of this Society, I drew attention, in a provisional way, to certain changes in the area of cardiac dulness and in the sounds of the heart which I had observed in association with signs of gastric ulcer. I promised then to give some day a fuller statement of these observations, and will attempt to redeem the promise to-night. I shall probably place what has interested me very greatly before you best by giving you the notes of a fairly illustrative case, abbreviated as far as possible.

CASE.—F. B., a woman, aged 28, and a cook by occupation, was admitted to Charity Ward, St. Thomas's Hospital, on October 11th, 1889.

Family History.—Her father was alive and healthy. Her mother, one brother, and one sister had died of consumption.

Previous History.—Had been generally strong and healthy, though always pale. Had never had any serious illness, and had never had rheumatism. For nine years she had suffered from "indigestion," consisting in pain after food, a sense of fulness and discomfort over the stomach, and pain between the shoulders. With this there was no vomiting, and the appetite did not fail.

Present Illness.—On September 11th, 1889, that is to say, a month before admission, she was employed in moving heavy weights. She felt none the worse for this until the next morning, when she vomited "a quart" of dark blood, and was afterwards greatly prostrated. During the next fortnight she vomited all food, and with it generally a small quantity of blood. She was fed by nutrient enemata. During the week before admission she vomited liquids, but not solids, and had no further hæmorrhage. The catamenia had been regular, always profuse.

On admission she presented the appearance usually associated with gastric ulcer in a young woman. She was anæmic but not emaciated; her skin was plump and rather transparent; the lips and conjunctivæ were very pale, and she complained of extreme weakness. Her appetite was fairly good, and she had now no vomiting nor pain after meals.

Abdomen.—There was neither distension nor contraction. No tumour could be felt, but there was localised rigidity of the muscles of the wall from the umbilicus to the xiphoid cartilage. She complained of a feeling of soreness on light pressure over the epigastrium, increasing to acute tenderness on firmer pressure. The tenderness could not be localised to any particular spot. There was no enlargement of liver or spleen.

Chest.—The chest was well formed and moved well.

Heart.—On percussion the upward dulness was found to begin at the lower border of the third left costal cartilage, extending obliquely downwards and outwards to the left nipple line, and on the right to the right edge of the sternum. The impulse was greatly diffused, and was most marked in the fourth space on the left side half an inch interval to the nipple line. It extended in a wavy manner over the third and fourth spaces. On auscultation a soft blowing systolic murmur was heard at the point of chief impulse, and was conducted into the axilla. The second sound was reduplicated. Over the pulmonic area a blowing systolic murmur was heard conducted upwards. The second sound was accentuated and reduplicated. Over the third and fourth left spaces, between the costal cartilages, a harsh rubbing murmur accompanied without replacing the first, and to some extent, the second, cardiac sounds. No murmurs were heard over the aortic area.

Lungs.—Apparently normal.

There were no affections of the nervous system, and the reflexes were normal.

The eyes examined with the ophthalmoscope presented no affections of the optic discs or retinae.

The blood looked pale when shed; under the microscope the red corpuscles appeared to be diminished in number, and were mainly well formed; but a few were oval, and the rouleaux were not good. The white corpuscles were slightly increased in number.

The patient was kept in bed, and was placed upon milk diet, to which beef tea and four ounces of wine were added after a few days. She was ordered to have gr. xx of the saccharated carbonate of iron three times a day.

Progress of the Case.—She had no pain after food, or vomiting during her stay in hospital, and she steadily retained her colour; but meanwhile the dulness on percussion increased. On October 23rd, twelve days after admission, it had extended to the second left space, an inch higher than on admission. The rubbing sounds had disappeared, and all the valve sounds had become indistinct. There was still tenderness in the epigastrium, and still rigidity of the muscles of the abdominal wall in that region. A week later her skin had become more pink, and she felt better and stronger. The dulness had receded three quarters of an inch, and the impulse was much less diffused. The murmurs were present but fainter. On November 11th her colour had further improved, the upward dulness over the heart had receded to the upper border of the fourth left costal cartilage, the impulse to the lower part of the fourth space 1 inch internal to the nipple line. The first sound at the impulse was prolonged, and faintly murmurous; the second pulmonic sound was slightly accentuated; the rubbing sound over the third left space was again audible. No further change was observed in the conditions of the heart.

She was discharged on November 23rd apparently in very fair health; the blood was then normal in appearance. The temperature was normal throughout, excepting for a few hours after admission, when it rose to 100° F.

I have notes of sixteen similar cases occurring in my wards at St. Thomas's Hospital, which have been carefully abstracted by Mr. T. P. Cowen, lately my house physician. Several of these

enable me to add a record of accessory and instructive symptoms. I may say that all of them appeared to be suffering from gastric ulcer. They were all young women, all anæmic, and mostly not emaciated. It is hardly necessary in this Society to go deeply into the question of the diagnosis of gastric ulcer, but I suppose that we may take it generally that the existence of this disease is indicated by pain occurring in the region of the stomach at various intervals after food, and lasting for some time, by vomiting, usually relieving the pain; by hæmatemesis, most commonly at long intervals and in considerable quantity; by tenderness in the epigastrium, often limited to a small area; and by rigidity of the abdominal muscles over the seat of tenderness. The absence of any sign of tumour is, of course, a necessary part of the diagnosis.

To proceed next to an estimation of the meaning of the cardiac symptoms. At first sight they are, in this case, and in the others recorded, exceedingly like those observed in the endo- and pericarditis of acute rheumatism and chorea. We note the extension of the cardiac dulness upward on the left side of the sternum to limits reaching as high as the second left costal cartilage, or even above it; we note that while the transverse extent of this dulness at its uppermost point is not more than an inch or an inch and a half, it spreads outwards, as followed downwards, taking an oblique line from the outer part of the higher left costal cartilages, which is continued either to the left nipple or to the left of it. There is also extension of the dulness to the right, less easily determined by reason of the resonance of the sternum, but to be made out by careful percussion. We note also the shifting of the impulse to the left and somewhat upwards. We note again the changes in the quality and distribution of the impulse. Instead of being confined to a limited area, it extends in all directions, but more particularly upwards, being both seen and felt in the third space and even above it. The occurrence of friction sounds, more particularly recognised over the upper part of the dull area, may, I admit, be open to question. In this case and in others I firmly believe that I have heard them, and have demonstrated them to friends and pupils accompanying me in my visits to the wards. To my hearing two sets of sounds have distinctly offered themselves. I could hear plainly at and a little below the pulmonic area the endocardial sounds. In addition to these I could hear

sounds of a totally different character, and not altogether corresponding in time. They were soft murmurs, mainly audible towards the end of systole and the beginning of diastole. Putting these phenomena together, I have been inclined to believe in the existence of a pericardial inflammation, producing roughening of the surfaces and effusion. The existence of a mitral systolic murmur, conducted into the axilla, and balanced by a marked accentuation of the second sound over the pulmonic area, has appeared to me to lend ground to the diagnosis of the existence of endocarditis, like that observed in acute rheumatism. So far as pathological observation in acute rheumatism can form a basis, this combination of symptoms would, I think, be generally accepted in any case, as proving the existence of endo- and pericarditis, but in the cases which we are considering there is no arthritic affection, and, what is more important, no pyrexia. I have therefore to ask myself and you how far it is possible that the lesions of peri- and endo-carditis may occur without pyrexia. Here I am confronted by a very serious chasm. I have no *post-mortem* examination of any such case to record, and can only work upon what may be called parallel lines. It is incumbent on me, therefore, to search carefully for other possible explanation of the phenomena which I have been putting before you. It appears to me to be worthy of consideration whether, in feeble and anæmic persons, the left lung may recede upward, and leave the heart in larger contact than the average with the chest wall. Such shrinkage would certainly increase the upward extension of dulness, and would favour the upward movement of the impulse. Probably, also, it would favour the tilt of the impulse to the left. It would also allow of the greater distribution of the impulse, but would hardly, I think, give rise to friction sounds apart from alterations in the pericardium. The shrinkage of the lung would involve less than the normal pressure of the heart and pericardium against the chest wall. An increased pressure would rather be required for the production of exocardial murmurs. It appears to me improbable that a diminished pressure would have such an effect. When I turn to the endocardial murmurs, difficulties worthy of solution arise. The patients are anæmic, and in anæmic people murmurs are notoriously often heard, replacing at one or other position the normal sounds. Such murmurs are certainly most commonly to be heard over the aortic valve, where they are systolic in time. I

have certainly heard them also over the mitral valve, both in systole and diastole, as far as I can remember, whenever the case has been one of simple anæmia or debility, aortic as well as mitral murmur has been present, and in simple anæmia and debility the murmurs have been much more temporary and fugitive than in the case which I have quoted. Again, in simple anæmia, I have not noted the accentuation of the second pulmonary sound, markedly present in the case which I have described. Anæmia surely means imperfectly filled arteries, and, supposing the pulmonary artery to be ill-filled, one would not expect increase or accentuation of the second sound over its valves. It is right, however, to consider what possible interpretation of this sign may be attached to a diminution in the size of the lung. If the shrinking be due to defective inpour of blood through the pulmonary artery, the accentuation would be diminished rather than increased. But it is not to be regarded as impossible that, as a part of the nerve-disturbances of anæmia, there may be tension in the pulmonary artery at once diminishing the circulation in the lung and increasing pressure in the artery. A third possible point of view is that of the altered action of the heart, due to impaired nutrition. A left ventricle may be doing its work imperfectly, with the result that a right ventricle, itself also weak, becomes dilated. A large dilatation of this kind would certainly tend to a displacement of the impulse upwards, and, probably, outwards. Such a dilatation, by altering the general shape of the heart, might produce murmurs at valves, and would certainly have as its corollary accentuation of the second pulmonic sound. It would also be accompanied by transverse extension of the cardiac dulness; but from all one's experience of dilated heart in old and enfeebled persons, I am of opinion that it would not materially determine upward extension of the cardiac dulness, and would determine much more dulness to the right than I have observed in my cases. Lastly, I should like to consider how far localised changes in myocardium may determine endocardial murmurs. A few years ago I was present at the *post-mortem* examination of a patient who had died of pernicious anæmia, and who had presented a marked mitral regurgitant murmur. There was no sign of disease of the mitral valve, but there was a remarkable rigid prominence of the muscoli papillares belonging to the mitral valve. While the rest of the heart was flabby, they were rigid and rounded. On

section they were distinctly changed. In the centre of each was a yellowish area, surrounded by a fine line of a deep red colour, outside which the muscle appeared to be normal. The appearance altogether was that of an inflammation of the central portions of the papillæ. In much thinking over the whole subject I have deemed it possible that one explanation of the phenomena which I am laying before you might lie in the existence of a myocarditis, determined by an imperfect nutrition, extending by continuity to endocardium and pericardium, and determining incidentally by interference with the balance of muscular action within the heart the occurrence of murmurs. In the case which I have just quoted, for instance, rigid swelling of the muscoli papillares must have interfered effectively in preventing accurate closure of the mitral valve in systole.

Since the writing of this paper has been commenced, I have read the thesis of my friend Dr. A. Foxwell, published in the 'Lancet' of October 24th, 1891, in which certain changes observed in the heart in debility are carefully stated and considered. The changes are certainly in several ways comparable to those to which I am drawing attention. They consist in increase of the cardiac dulness, both vertical and horizontal, in tilting of the impulse to the left and upwards, in the occurrence of pulmonary and mitral murmurs. The interpretation is that in weak persons the right ventricle tends to become over-distended, that as a result of this the rest of the heart is thrust upwards, encroaching on the left lung and carrying the impulse upward and to the left. The upward extension of the cardiac dulness is recorded to be very considerable in some instances. It is clear that this paper must be taken into account when the meaning of the cardiac changes related with gastric ulcer is sought.

But it is not clear how far the cases upon which Dr. Foxwell's observations are founded correspond with those examined in the present communication. I am discussing simply cases of gastric ulcer, in which certain symptoms of cardiac change and disorder have been observed. Dr. Foxwell's observations present to us a new and very original view of cardiac changes occurring in debility. They appear to me to be worthy of careful regard and comparison. They may, indeed, go far to explain some of the difficulties to which I am introducing you, for it is clear that the whole matter is very complex.

Lastly, it must be borne in mind that in gastric ulcer the position of the heart in the vertical aspect is likely to be high. Noting in passing that tight lacing is very often to be taken into account, the fact of the existence of gastric ulcer involves a relatively high position of the diaphragm. Such a raised position of the diaphragm would presumably oppose downward movement of the heart if enlarged, and would involve also encroachment of the heart upon the lungs. We may contrast this with the heart dilated on the right side in cases of emphysema, where the enlarged lung tends to push the heart downward and the exaggerated action of the diaphragm, compensative of diminished movement of the chest walls, readily allows, or even facilitates, the downward movement of the heart.

On review of all the symptoms presented in the case first quoted, and the others of which the notes are presented, I am inclined to think that the hypothesis of the occurrence of trophic changes in the endo- and peri-cardium has much to be said in its favour. Admitting the absence of proof derivable from autopsies, I am inclined to believe that we find evidence, from the clinical side, of the occurrence of endo- and peri-carditis, unaccompanied by fever, in young women suffering from gastric ulcer and anæmia, particularly where the anæmia has been suddenly produced or exaggerate by hæmorrhage. I must pray you to let me think out before you the speculations suggested by this aspect of the cases. Much has been written with regard to the causation of gastric ulcer, but little certain conclusion has been arrived at. The round or oval perforating ulcer is neither in form or character such as the ulcers which one finds in chronic gastritis. That it is due to blocking of a small artery by embolus or thrombosis is, so far as I know, not proved, although the suggestion that the limited area of mucous membrane supplied by such an artery would be exposed as practically dead matter to the action of the gastric juice is ingenious and attractive. The circular form of the ulcer would, as far as I know anything of the minute distribution of arteries towards the surface, not support this view. The outline should be rather sinuous or indented.

If we look to the skin for purpose of comparison, the circular forms of ulceration most frequently met with are rupial, herpetic, and bullous. With the first of these we have not here to deal. In the other two we can certainly recognise the result of perverted

nerve influence as affecting nutrition. Now, in the cases with which we are dealing, herpes of the mouth and herpes in the conjunctiva are far from uncommon. I have no record of the occurrence of bullæ, although I have asked carefully of my patients on this point. Nevertheless, I think it fair to suggest that the ulcer in the stomach may be due to altered trophic influence of nerves. You will remember that the cases under consideration are in all but few instances those of young women, anæmic and yet apparently well nourished. It appears to me that the appearance of good nourishment is itself fallacious. The skin, as compared with the skin of health, is too transparent, too resistant to the touch, and too thick, as far as tactile examination can indicate. With all this appearance of delicacy it has a yellowish tinge, which is not that of health, is certainly not one of simple bloodlessness. I think you will all agree with me in recognising an absolute difference between this skin and the skin of a woman who has lost blood in large quantities from other sources than the stomach, and with other association of symptoms than those of gastric ulcer. I think that we may see here the existence of changes of a specialised kind. In the class of patients under observation, functional disorders in the area of distribution of the pneumo-gastric nerve are undoubtedly common. I have been led to think whether these may advance to trophic disturbance within the same area. Going a step further, and accepting the existence of gastric ulcer in the cases, we may approach the meaning of the heart disturbance. One might argue as to the possibility of this being determined by reflex influence passing to the centre from the stomach and outward again through the cardiac nerves. My general experience of similar conditions would hardly lead me to think this very probable. To take herpes. In most, if not all, of the occurrences of herpes I have found it secondary rather than primary. The labial herpes of pneumonia and gastritis, the preputial herpes of prostatic congestion, the oral herpes of dyspepsia and uterine irritation are instances in point. I cannot forget the fact that in a large number of the cases under consideration uterine disorder exists, and may possibly, in a reflex way, give rise to the gastric ulcer; but, on full examination, I dare not put forward the idea that a gastric ulcer is *per se* capable of exciting serious trophic disturbances in a large viscus like the heart. Therefore, I am impelled to take into consideration the possibility that both the gastric ulcer and the

cardiac changes may be common results of a pneumo-gastric disturbance. Even as regards acute rheumatism, there is not a little to support the idea that the cardiac inflammations may be referred to perverted pneumo-gastric influence. I heard an excellent observer, Dr. Buzzard, say, at one of the Societies, that he was incited to think whether many of the phenomena of acute rheumatism might be referred to the medulla oblongata, citing, in addition to the affection of the joints, the affections of the heart and the febrile perspirations. Is it possible that in an anæmia such as we are discussing this most sensitive nerve-centre may be set into dystrophic action?

It is well known that a form of optic neuritis is found in cases of anæmia; I do not know how far in cases of the anæmia of gastric ulcer as compared with others. In one of my cases optic neuritis was present for a considerable period. I have the authority of Mr. Nettleship for this statement. In other cases, and, indeed, in the case in which optic neuritis was observed, marked signs of inflammatory or hyperæsthetic affections of serous membrane have been noted. These include symptoms of meningitis, cerebral and spinal, of pleurisy, and of peritonitis. In one case, particularly, where no tubercle could be detected, a most painful peritonitis with effusion lasted for many weeks, disappearing ultimately with the rest of the symptoms. Such serous inflammations have their importance when we are considering affinities with acute rheumatism. Still greater becomes their importance when we find that no other kinds of visceral lesion, save those of the heart, have to be chronicled.

To sum up, in presenting this paper I am desirous of drawing your attention to certain phenomena which, so far as I know, have not been stated before, and of which I am at present unable to give a certain explanation. I present the cases in the hope that others may, where they have opportunity, investigate the phenomena and subject them to criticism. It is a point of the greatest importance that I have had no opportunity of verification by means of *post-mortem* examination, and that, to a certain extent, my suggestions are hypothetical. I think, however, that everybody will allow that the diagnosis of pericardial effusion and of endocarditis is daily made and admitted in connection with acute rheumatism. Such diagnosis rests on well-observed pathological conditions and on experiment. The physical signs noted in the

cases presented appear to me to correspond very completely with the physical signs accepted as proofs of the existence of such pericardial effusion and endocarditis, but they have attached to them, besides the fact of their not being demonstrated pathologically, the extremely important fact of the absence of pyrexia. How far it is possible that the absence of pyrexia may be explained by the particular kind of anæmia exhibited by persons suffering from gastric ulcer, I am unable at present to say. In conclusion, I trust that the Society will accept my communication as a record of observations in respect of which full explanation is still to be sought.

APPENDIX.

Of the 17 cases on which this paper is founded, 16 were female, 1 male. Of the female cases, 5 suffered from very marked anæmia, 8 were noted as "anæmic," 1 as "somewhat anæmic," and in one case there is no note of this condition. The catamenia in 6 cases were regular, and in 2 of these were noted as profuse. In 6 cases they were irregular, and in 1 case they were recorded as occurring every fortnight. In only 2 out of the 17 cases had the patient suffered from acute rheumatism. All the cases presented, more or less, the symptoms of gastric ulcer. All had pain after food, and all but one had vomiting, there being marked nausea in the exceptional case. There was tenderness localised in the epigastrium in 14 cases, somewhat diffuse abdominal tenderness in one. There was hæmatemesis in 9 cases, in the remaining 8 it was noted as absent. Melæna was noted present in 3 cases.

With regard to the cardiac symptoms, dulness commenced above at the level of the third left costal cartilage in 8 cases, in the third left interspace in 1 case, at the fourth cartilage or rib in 4 cases, at the fifth in 1 (the man), was described as normal in 2, and was absent in 1. The impulse was in 4 cases described as in the fourth space just internal to the nipple line, in 1 case as in the fourth space 1 inch internal to the nipple line, in 4 cases in the fifth space just internal to the nipple line, in 1 as in the fifth space 1 inch below and internal to nipple, in 1 in the fifth space in the nipple line, and in 1 as $1\frac{1}{2}$ inches below nipple and just internal to the nipple line. In 5 cases it was described as normal.

In 14 cases a murmur was heard at the apex, conducted into the axilla, and in 1 case on admission a doubtful murmur was heard

in this position. An undoubted murmur afterwards developed in this case during her stay in hospital. In all these cases there was accentuation of the second sound over the pulmonary area. A presystolic mitral murmur existed in 1 case. In 7 cases a systolic murmur was heard over the pulmonary, and in 4 over the aortic, valves. To-and-fro friction murmurs along the left border of the sternum were heard in 7 cases.

With regard to temperature, it was raised on admission in 2 cases, viz., to 99.4° and 100° respectively. In the former case it rose subsequently on one occasion only to 100° . In none of the other cases was there any rise of temperature, either at the time of admission or afterwards.

Dr. SANSOM said that the interesting paper of Dr. Ord clearly established a close association between the cardiac and the gastric signs, which he described in his cases. It appeared to him that it was shown that in these enlargement of the heart coexisted with gastric disturbance giving rise to symptoms resembling those of ulcer of the stomach, though it was not proved that they were identical therewith. The diagnosis of enlargement of the heart was based upon the physical signs. (1.) The outline of the chamber, both right and left, determined by percussion, was greater than the normal. It could scarcely be contended that such enlargement of the area of dulness could be due to pericardial effusion, at any rate in all the cases. (2.) The occurrence of the so-called reduplication of the second sound, in his (Dr. Sansom's) opinion told in favour of dilatation of the left ventricle. The more he observed this phenomenon, the more he felt convinced that it was a simulated and not a real doubling of the second sound that was heard. It was to be ascribed, just as the triple or cantering rhythm, the "bruit de galop," to the sudden entry of a wave of blood into a comparatively toneless ventricle. This, as an explanation of the "bruit de galop," was given by Potain and confirmed by the careful cardiographic investigations of François Franck. He believed that the so-called reduplication of the second sound was never due to want of synchronism in the closure of the valves of the aorta and pulmonary artery respectively, as so generally supposed. He took, therefore, the sign in Dr. Ord's cases to signify a temporary dilatation of the left ventricle. (3.) The murmur, systolic in tone, at the apex might be due to other causes than endocarditis. It was evanescent, and might be placed in the same category with those observed not only in anæmia but in neurotic disturbances of the heart in the absence of anæmia. Dr. Ord's observation of actual changes in the summits of the papillary muscles in a case of pernicious anæmia was very interesting: it was most probable that degenerative and other changes would occur inferentially in these terminal structures, and would impair the due coaptation of the mitral curtains in systole. (4.) The sound having the character of pericardial friction, heard over a limited area and disappearing without the usual signs of adhesive pericarditis, was very difficult of interpretation. He would hesitate long before ascribing it to effusion of inflammatory products, seeing that there were no evidences of rheumatism nor of any of the usual associations, and no concomitant fever. Murmurs of cardio-vascular vibration might be very

harsh and scraping, resembling the peculiar scraping sound of the subclavian murmur, and yet be quite independent of pericarditis. On the whole, he considered the signs showed a temporary dilatation of the heart, and if asked if he had observed such cases—a sort of general congestion of the heart as it were—he could answer “Yes.” He could not agree that such dilatation was in causal relation with anæmia. In some cases presenting the most pronounced murmurs, associated with anæmia, the heart was small ; but he had observed a general enlargement of the heart, temporary and passing away without manifestation of special physical signs, without any manifestation of anæmia. The evidence of association in Dr. Ord’s cases with derangement of the stomach was clear. There was undoubtedly hæmatemesis in one case, but it did not appear to him that in all we should be justified in the diagnosis of gastric ulcer. There was great force in the argument that the central nervous mechanism was in fault in these cases, that the pathogenesis of the one was the pathogenesis of the other.

Dr. ROUTH said that Dr. Ord had stated that in many cases it was impossible to diagnose ulcer of the stomach from mere functional dyspepsia. This might be so, but Dr. Ord had not referred to *electricity* as a means of diagnosis. A patient might have sickness vomiting, even sometimes by reason of its severity vomiting of blood, and great pain after food, and yet it might only be functional. Now in these cases there was great pain also in pressing over the epigastrium, so as sometimes to make the patient scream. Now if electricity, the interrupted or continuous currents, were applied on both sides of the neck over the vagus nerve, in about ten minutes all pain disappeared at the epigastrium, and you could press as hard as you liked without troubling the patient, and she would go home and eat a good dinner without pain. This he had seen Apostols demonstrate several times, and had done it himself. Sometimes one pole could be placed on the stomach itself with better advantage. If it were an ulcer, on passing the current over the stomach, especially the positive pole, pain would be intensified at the spot where the ulcer was. Then he thought that the enlargement of the heart, temporary though it was, would itself irrespectively of endo- or peri-carditis, give rise to murmurs and friction sounds, the latter, however, generally being single. His old master, Dr. C. J. B. Williams, had demonstrated this often at the bed-side. After rheumatic endocarditis and pericarditis, when the patient was quite well again, and convalescent, and all sound had disappeared, if the patient over exerted himself, or over ate, or over excited himself, the heart would be found to enlarge, and occupy a much larger surface, producing the “*engorgement*” of the French. This could be very easily made out by percussion, and then you would hear for a day, or half a day, cardiac murmurs, mitral or aortic, reduplications, &c., and single rubbing sounds—all of which, as the heart resumed its normal size, as rapidly disappeared. He thought, therefore, these cases favoured the view that the murmurs were entirely due to temporary enlargement, and perhaps wrinkling of some parts of the pericardium, and not due to inflammation.

The PRESIDENT thought that in the first case which had been related there was no escape from the diagnosis of pericarditis. The whole of the physical signs, indeed, pointed clearly to that conclusion. The absence of pyrexia was perhaps not such a difficult matter to explain, for it was well known that the effect of hæmorrhage was to reduce a normal temperature and to depress considerably a pyrexial one. In cases of hyperpyrexia he had often heard a superficial friction sound over the heart, but

it was transient, and seemed to be due simply to the pericardium being unduly dry; he would not think of calling such a sound pericarditic unless it persisted after the temperature fell. He had never heard such sounds in association with moderate fever or with apyrexia. With regard to the endocardial murmurs, he felt a doubt if they were due to organic change. As to the underlying cause of the gastric ulcer and the cardiac symptoms, he found a difficulty in accepting the neurotic theory. He thought that a neurosis would not quite explain the profound and long-standing anæmia so often accompanying these cases, and also the painful digestive symptoms. He had looked upon the ulcers as primarily originating in dyspeptic abrasions in the stomach, such as one frequently saw in the buccal mucous membrane, or such as occurred in the œsophagus. Such an abrasion would expose the deeper parts to uncontrolled action of the gastric juice, and thus the lesion could be produced.

Dr. WHEATON said that having had the opportunity of examining many of Dr. Ord's cases, he could confirm his statement that the physical signs of endo- and peri-carditis were present. For the last three years he had been on the look-out for pathological evidence bearing on the subject, and mentioned one case. The patient, a servant at the Waterloo Road Hospital, was a healthy-looking but rather anæmic girl, aged 18. One evening, after partaking of a glass of milk, she was seized with symptoms of peritonitis, and admitted into the wards under the care of Dr. Haig. On admission there were signs of intense peritonitis and great collapse, a blowing systolic murmur was heard along the left side of the sternum in the third and fourth intercostal spaces; and the second sound at the left base of the heart was accentuated. The patient died three days later. At the autopsy, intense general peritonitis was found, and two ulcers, one on the anterior and the other on the posterior surface of the stomach, both of which had perforated, probably simultaneously. The ulcers had the usual punched-out appearance, and were each about the size of a shilling. On opening the chest, the heart was seen to be greatly dilated, there was no pericarditis, the pericardium contained two and a half ounces of clear fluid. The left ventricle was much dilated, and the edges of the mitral valve acutely hyperæmic and swollen, with small nodules scattered on its auricular surface. The cordæ tendinæ of the valve were also distinctly shortened, and its orifice contracted. Microscopic examination of the mitral valve, confirmed the appearances of acute endocarditis. The patient had never had any vomiting, hæmatemesis, melæna, or rheumatism; and at the time of her illness was performing her usual duties at the hospital. He expressed a hope that further pathological evidence would be obtained to elucidate Dr. Ord's paper.

Dr. ORD, in reply, said that in all the cases the signs of gastric ulcer were present, but they were more marked where there was recent or severe hæmorrhage. He did not positively assert that either peri- or endo-carditis was present, but he held that it seemed to be a fair inference. As to the question of limited pericarditis in cases of enlarged heart, it might only be heard at the spot where the auricle overlapped the ventricle. He was still disposed to believe that the reduplication of the second sound heard over the pulmonic valve was real in both cardiac and renal disease. He wondered if anæmia could be the common cause of both gastric ulcer and cardiac affection. It might act just as starvation did in producing ulcer of the cornea.

A CASE OF RAYNAUD'S DISEASE, WITH PAR- OXYSMAL HÆMOGLOBINURIA.

By A. HAIG, M.A., M.D., Oxon., F.R.C.P.

ELIZA H——, aged 6. Admitted under my care in the Royal Hospital for Children and Women, November 27th, 1890.

Her family have always lived in London. There are three brothers and sisters younger and one older than the patient. Mother has had nine children born alive, no miscarriages.

Mother is subject to cough.

Mother's mother and mother's brother had rheumatic fever.

Father's mother had "chalk gout."

No ague in family.

Patient had measles and bronchitis when 15 months old, and whooping cough at 4 years old. Eyelids and face generally very pale and puffy. Has conjunctivitis and opacity of corneæ.

On admission—pulse 120, temperature 100°, respiration 24.

Soon after admission, she passed some urine which was almost black, threw down a grumous deposit, and contained a large amount of albumen. A few hours later it was the colour of dark sherry, and, later, still quite normal.

December 1st. Urine to-day amber clear, acid, 1018. Phosphates. No albumen. I examined her heart and found reduplication of the first sound between the apex and the left border of the sternum, and a loud second sound both at apex and base.

Pulse quick, 80 to 105, irregular in force and time; and it was difficult to judge by the finger as to the tension; but pulse traces showed at times considerable tension. When the pulse was quick, I think probably there was some failure of the heart, and tension was not well maintained on this account.

Spleen dulness normal.

The urine of November 28th was port wine-coloured, with a grumous sediment 1018. Contained a very large amount of albumen (about 10 or 12 parts per 1000, Esbach). Gave hæmoglobin lines with the spectroscope and a relation of uric acid to urea of 1 to 30.

December 2nd. "The child's hands were washed with cold water at 6 A.M. After this she was well till 1 P.M., when she began to

complain of pain in the hands, which were seen to be quite blue up to the level of the wrist. This blueness persisted up to 1.10 P.M., when the colour only remained beneath the finger nails; the rest of the hands were red, puffy, and markedly swollen, but quite cold; the puffiness and redness gradually faded, and at 2.30 P.M. the hands were normal in appearance and temperature. Pulse 80, no marked plus tension during the stage of blueness and congestion. Heart first sound prolonged, second sound accentuated at apex and base. The child had marked pallor and appeared to have intense headache during the attack in the hands; but said she did not know what was the matter, and had no headache. At 2.30 P.M. she sat up and began to play with her toys.

"Blood drawn from finger as congestion stage was passing off showed marked excess of white cells, which were actively motile and putting out many processes. Red corpuscles formed bad rouleaux and scattered among them were collections of bright refracting nuclei about the size of one-sixth of a red cell, and it appeared as if in places the cells had burst and discharged their contents." I am indebted for the above description of an attack and the condition of the blood to the notes of my colleague, Dr. S. W. Wheaton, who was then Registrar.

December 4th. Urine of twenty-four hours, ending this morning, during which there was no attack, was 19 ozs., amber smoky, turbid 1020, faintly acid. No albumen, some phosphates, uric acid, urea relation 1—17.

Had attacks of local asphyxia in hands or feet, or both, on December 6th, 7th, 8th, 9th. On the 11th and 12th there were no attacks, in spite of her hands being washed in cold water as before. The above attacks lasted from ten to forty minutes, and attention was directed to them by the child complaining of pain in the affected members. They were not accompanied by any marked rise of temperature or followed by hæmoglobinuria, in fact, the temperature was sub-normal all the time.

December 13th. Urine of twenty-four hours, ending 8 A.M. to-day, 24 ozs., pale amber, turbid neutral, 1016. Urea, 1.2 per cent.; uric acid, 0.04704 per cent. Relation, 1 to 25. Weight, 38 lbs. Urea, 3.5 grains per pound, which, for a child of her age, is very low and shows failure of nutrition.

Had a slight attack in the right hand at 7.15 this morning, lasting ten minutes.

December 15th. Urine of twenty-four hours, 25 ozs., pale amber, slightly acid, 1019. Urea, 1·4 per cent.; uric acid, 0·05712 per cent. Relation, 1—25.

Urea still very low. She is on farinaceous diet, and sister says she is always very hungry (had she been starved?). I ordered her to have meat and be fed up. No doubt this low nutrition and relative excess of uric acid is the cause of all her trouble.

December 16th. Had four distinct attacks in hands and feet, first at 11.25 A.M.; second, 4.25 P.M.; third, 4.50 P.M.; and fourth, 5.30 P.M. Urine passed between 9 and 11 A.M. to-day, 2½ ozs., amber, turbid, neutral, 1025. Uric acid urea relation, 1—19.

Urine 11 A.M. to 1 P.M. (being a mixture of the urine corresponding to the attack at 11.25 A.M., lasting twenty minutes, with the urine of the remaining one hour and forty minutes free from attack), 1026, faintly acid; uric acid to urea, 1—23.

There was thus apparently less uric acid at the time of the attack than before it, but, as I have pointed out in the case of headache, epilepsy, and other paroxysmal effects of uric acid, there is a variation in the excretion in both directions, a plus excretion during the attack and a minus before or after it, or both, and when the separation is not accurate, as in slight attacks it can very rarely be, the minus excretion before or after the attack more than balances the plus excretion during the attack (which only lasted twenty minutes out of two hours urine), and we get the result here shown, namely, that there was a greater excretion in the two hours before the attack than in the two hours including the attack; in both periods, however, there was a considerable excess of uric acid, and the urine is, after all, but an imperfect index of the amount of uric acid in the blood.

December 19th. Had no attack on the 17th and 18th. To-day she had an attack at 1.50, lasting only ten minutes, but followed by a rise of temperature to 102·8°, and the urine passed next after this, at 5 P.M., contained hæmoglobin.

Urine passed at 1 P.M., before the attack, pale amber, slightly acid, 1026; uric acid to urea, 1—21.

Urine passed at 5 P.M. (the first after the attack), grumous, red-brown, with powdery sediment, strongly acid, 1030. Hæmoglobin lines. Much albumen, but part of the turbidity clears with heat (? urates). Uric acid urea relation, 1—22. Total urea in 100 c.c. was 49 grs. Total uric acid, 2·2 grs. Total acidity reckoned as

oxalic acid, 5·8 grains. Relation of acidity to urea, 1 to 8·4, that is to say, acidity was low, and with a low acidity we had a large excretion of uric acid, 1—22. But I said, above, the urine was strongly acid; how then, it may be asked, was the acidity low? I reply that the urine (as is generally the case with excess of uric acid in the blood-uric-acidæmia) was a very concentrated one, and the excretion of water small; hence the apparent acidity was high; but when we came to reckon the total acidity by the side of the total urea we find that the acidity is relatively low, for we only find 1 grain of oxalic acid for 8·4 grains of urea, whereas my researches show that taken over a long period of time, including 600 or 700 days, the relation of acidity to urea is 1 to 6·6: 1 grain of oxalic acid for every 6·6 grains of urea, whereas here we only got 1 grain for 8·4 grains of urea, so that, as I said, acidity is really low, though, estimated only with a piece of litmus paper, it would appear high. Such is the difference between exact and inexact investigation, a difference, I regret to say, not always appreciated.

What really happened in the attack of hæmoglobinuria was, I have no doubt, somewhat as follows:—At the time of the local asphyxia, 1.50 P.M., there was intense uric acidæmia and an enormous excretion of uric acid in the urine, having the relation, say, 1—8 or 1—12 (a relation which I have actually found in other cases of the disease where the separation of urine, in accordance with the attack has been more perfect),* but in this case the attack only lasted for ten minutes, and the excessive excretion only went on probably for half an hour, but we unfortunately got no urine passed till 5 P.M., nearly three hours later. Meanwhile the temperature has risen, the alkalinity of the blood has diminished, the excretion of uric acid has fallen greatly, possibly down to 1—40 or 1—50, showing that there is no longer any excess of it in the blood, and the acidity of the urine has risen (it was possibly alkaline at the time of the attack), but the blood has been flooded with hæmoglobin, and this slowly finds its way out of the kidney. The result is that in the urine passed at 5 P.M. we have plenty of hæmoglobin, a low acidity, a large excretion of uric acid, 1—22, though this, in all probability, was the product of a mixture of the very much larger excretion during the attack with the very much smaller in the 2½ hours that followed it, so that the relation of uric

* See 'Uric Acid as a Factor in the Causation of Disease.' J. and A. Churchill, 1892.

acid is reduced from 1—8 to 1—22; just as the acidity is a mixture of the alkaline urine of the attack with the highly acid urine of the $2\frac{1}{2}$ hours following it, when with high temperature there would certainly be a rise of urinary acidity and a corresponding fall in the alkalinity of the blood.

Now that I have gone into these matters at length, I shall not require to repeat the explanation of the same phenomena in subsequent attacks.

Dr. Wheaton examined the blood one hour before an attack, and found 3,800,000 red cells per cubic millimeter; he again examined it one hour after an attack during hæmoglobinuria and found 2,990,000 cells only, a very great diminution. He repeated the examination on two subsequent occasions with similar results.

December 20th. She had an attack of local asphyxia in both feet, lasting from 6.55 to 7.30, and followed by a rise of temperature to $99^{\circ}8'$. The urine passed after this, at 8 P.M., contained hæmoglobin.

Some urine passed before (? how long) the attack gave a relation of uric acid to urea 1 to 28, and that passed at 8 P.M., containing hæmoglobin, gave a relation 1 to 30. Here, again, I have no doubt there was an admixture of before or after with during the attack, but as I was unable to obtain definite information as to the hour at which the specimen before the attack was passed, no valid conclusion can be drawn from my results. I got many such results with the uric acid headache before I found out the importance of separating carefully the urine of the attack.

December 23rd. Local asphyxia toes of both feet 3.20 to 3.35 P.M., or lasting slightly till 4 P.M. Urine passed during the attack of a pale port-wine colour (a mixture of before and during), slightly turbid, alkaline, 1022; uric acid to urea 1 to 23.

Urine drawn by catheter at 4 P.M. (the end of the attack, excretion of about half an hour) dark port-wine colour, brownish deposit, slightly acid; uric acid to urea 1 to 28.

The remarks I made before about the effects of admixture apply here. Also, the first urine was no doubt a mixture of before and during or the relative amount of uric acid would have been greater, and that drawn by catheter is really the excretion of a period after the attack, when the blood is almost clear of uric acid, and its effects are passing off.

On December 21st she passed some hæmoglobin at 6.15 P.M., again on the 24th at 1 P.M., and on the 25th at 9.30 A.M.; in none

of these cases was the passage of the hæmoglobin apparently preceded by local asphyxia; possibly the attack escaped observation; the child was very heavy and dull in the attacks, and if other children were making a noise her first cry of pain might not have been heard. Later, on December 25th, however, 5.15 P.M., both feet were affected, and remained so for about an hour, and the urine passed at 6 P.M. contained hæmoglobin, as also the specimen next passed at 6.15 A.M. on the 26th, but that at 9 A.M. on the 26th was clear.

December 28th. She had an attack in which the left foot led the way, and was followed by the right from 5.15 to 7.30 P.M.

December 29th. The feet and hands were both affected from 5 P.M. to 6 P.M., and the temperature rose to 100.8° , but the attack was not followed by hæmoglobinuria.

December 30th. The hands and feet were affected from 3 to 3.45 P.M., and again from 5 to 8.30 P.M. The urine passed at 8.30 P.M. contained hæmoglobin, and that at 9.30 the same.

I did not estimate the uric acid urea relations in any of these specimens, because it did not appear to me that the separation was sufficiently accurate to make it worth while to do so; the attacks were slight, lasting from only a few minutes to half an hour, and in no single instance was the urine corresponding to the attack obtained separately.

In a more severe case of Raynaud's disease, where an attack lasted nearly twenty-four hours, there was much less difficulty about obtaining the excretions corresponding to the attack, and I got, as I have said, a relation of 1 to 8. That is to say, with the more severe and prolonged attack there was a greater and more continuous excess of uric acid in the blood and urine.

December 31st. Hands and feet affected from 5.15 to 7.30 P.M.

1891. January 4th. Left hand affected from 3.25 to 4.10 P.M.

January 5th. Hands and feet „ „ 9.30 „ 10.45 P.M.

Right hand „ „ 1.5 „ 1.30 „

„ 6th. Hands affected from 1.30 to 2.30 P.M., and temperature rose to 99.4° .

„ 7th. Hands affected from 9.50 to 10.10 P.M.

Right hand affected from 5.10 to 5.35 P.M.

Both feet „ „ 6.45 „ 7.15 „

„ 9th. „ „ „ 6.45 „ 7.45 „

„ 14th. Both hands „ „ 6.5 „ 6.25 „

„ 15th. I examined the urine of twenty-four hours end-

ing 2 P.M. to-day, 18 ozs.; sp. gr. 1018, and found:—Urea, 1·7 per cent.; uric acid, 0·06384 per cent.; total urea, 161 grs.; uric acid, 5·2 grs.; acidity, 15·7 grs.; uric acid to urea, 1 to 27; acidity to urea, 1 to 9. This shows that urea is still very low per pound of body-weight for a child, acidity is low, and uric acid high; hence recurrent excess of uric acid in the blood, which is the cause of all our trouble.

She is up and going about, but seems dull and cross. Eyes still very bad.

January 17th. Feet affected from 12.5 to 12.45 P.M.

„ 18th. Both hands affected from 10.30 to 11.15 A.M.

Left hand „ „ 5.30 „ 6.30 P.M.

„ 19th. Feet and hands affected from 5.45 to 6.30 A.M.

Right hand affected from 5.10 to 6 P.M.

„ 20th. „ „ „ 6 to 7.15 A.M.

„ „ „ 11 to 11.30 A.M.

„ 22nd. Left hand „ „ 5.40 to 6.15 P.M.

On this day, as the attacks seemed to be pretty frequent, I determined to treat the uric acid, and she was accordingly put on salicylate of soda, gr. viij three times a day after meals, and had the first dose on the evening of January 22nd, after the forty-second attack of local asphyxia. She was also given a small dose of acid and strychnine three times a day to aid the action of the salicylate.

From this she went for twenty days right off without any attack whatever, her forty-third attack of local asphyxia occurring at 2 P.M. on February 11th while out on the roof of the hospital.

January 29th. Better and more cheerful, and all the nurses bear witness to the improvement in her temper, though her eyes are still very bad.

February 5th. Still no attack, and temper continues to improve. Yesterday, however, the temperature rose to 101·8° at 10 P.M., but there was no sign of an attack in limbs or urine.

February 11th. Attack while on roof at 2 P.M., part affected not stated.

February 13th. Feet and hands affected 11 A.M. to 1.30 P.M.; was out on the roof early in the morning.

February 14th. Seems well in herself, and is singing away. Salicylate mixture to be given four times a day.

February 19th. No attacks; has been on the roof twice for about an hour each time. Going on well.

February 22nd. She had her forty-fifth and last attack in both ears from 7 to 8.30 A.M., and it is noted that her temper was very bad at the time of the attack.

February 23rd. Temper is much better now than before salicylate was begun.

March 5th. Still no attacks in spite of exposure on the roof in the cold of the morning at 7 A.M. All drugs left off to-day.

She was on the roof on February 17th, 18th, 25th, and 27th without any effect being produced.

March 9th. Weather very cold with a strong east wind, so salicylate was resumed again.

March 12th. Was on the roof at 7 A.M. to-day. Thermometer 27° F. No effect.

She now went on quite well, her temper keeping placid, and she seemed bright and happy in spite of the condition of the eyes, which remain so bad that she can only see very indistinctly, and is afraid to walk or run by herself.

May 7th. Well in herself now, but had an attack of tonsilitis about two weeks ago, for which, as another child in the ward had diphtheria, she was isolated; she, however, got quite well, and had no sequelæ.

About a week ago I put her on small does of liq. calcis chlor. with the view of trying whether I could produce some retention of uric acid, and then, by bringing it out into the blood again, produce an attack. I, therefore, examined the urine of twenty-four hours ending this morning, and found total urea 187 grs., uric acid 7 grs. Relation 1 to 26. Acidity was still low, but urea had risen somewhat in accordance with the improvement in the child's general condition, being now about 4.6 grs. to the pound. It seemed, however, that with this continued low acidity I was not likely to produce much retention of uric acid, and I therefore gave up the attempt, and sent the child home.

June 22nd. She still attends as an out-patient; is cheerful and well, getting fat, and has had no attack of any sort.

She had thus altogether 45 attacks of local asphyxia and 9 of hæmoglobinuria; 42 out of the 45 attacks occurred in 52 days from December 2nd, 1890, to January 22nd, 1891, and 3 attacks in 31 days, from January 23rd to February 22nd, 1891. In the week ending January 22nd, the day on which salicylates were begun, she had 8 attacks, and then she went, as I have said, 20

days without an attack at all; then came 3 attacks in the next 11 days, and after that none, in spite of cold weather (as our winter last year certainly did not end on February 22nd) and exposure on the roof as before.

The points of greatest interest to me are the pallor and headache which occurred during an attack, as described by Dr. Wheaton. The fact that her temper was generally bad, and was worse in the attacks; also that it improved when salicylates were given, to such a marked extent as to be noticed by all who saw her.

The case also illustrates fairly well the fact observed by others, that the attacks are generally in the morning; and this corresponds with the well-known fact, that the greatest excretion of uric acid in the urine, and probably the greatest uric acidæmia, occurs in the alkaline tide of the morning.

Dr. Wheaton says that the pulse during an attack was 80, and showed no marked plus tension.

But 80 is a somewhat slow pulse for a child of 6 years. The quickest pulse on her charts is 142, and the slowest 54, and pulses of 60, 64, and 72 on several occasions corresponded with attacks, and these show considerable bradycardia, which, as I have argued, is due to acidæmia.

As to tension, I found that it was very difficult to estimate by the finger in this case; but I pass round some traces I obtained, and one of these shows considerable tension though the pulse rate was 80.

Again, I think that when her pulse was quick it was not always because the tension was low, for there were several signs of heart failure here, and, as I know from observations on other cases, when the heart fails before high tension its systoles become imperfect (as some of my traces from this case show) and its action quick; and Marey's law that pulse rate is inversely as arterial tension, or that the length of the ventricular systole is directly proportional to the amount of arterial resistance, holds only so long as the heart is equal to its work; and when the systoles become imperfect and the heart quickens, tension is not maintained well in spite of great peripheral resistance and contraction of arterioles. "A long pulse means a labouring heart,"* but a short pulse with peripheral resistance means a failing and dilating heart.

* Mahomed, 'Guy's Hospital Reports,' 1879.

Now comes the question of *post* or *propter*, always a difficult one to answer, and doubly difficult with regard to a single case. Was the wonderful improvement of the 22nd of January onwards a mere accident or was it part of a steady general improvement which happened to take more definite shape just at the time a salicylate was given; or was the salicylate the cause, the actual and determining cause, of the change in the aspect of the case?

From my point of view this is by no means a single and solitary case, as my theory is, as will appear from previous remarks, that all the signs and symptoms above described are mere exaggerations of conditions which are met with every day in physiology and pathology, and many of which I can produce, remove, or alter at pleasure.

The two main points of the case are: (1) the local asphyxia due to spasm of certain vessels (Raynaud), and (2) the paroxysmal hæmoglobinuria. With regard to (1) I have suggested in the 'British Medical Journal' that, *cæteris paribus*, arterial tension varies with the amount of uric acid that is circulating in the blood ('British Medical Journal,' February, 1889, p. 288), that is to say, that excess of uric acid in the blood contracts the arterioles and raises arterial tension, and I have shown ('Brain,' Spring Number, 1891, and elsewhere) that certain headaches and fits of epilepsy are probably due to its causing spasm in the vessels of certain vascular areas. If, then, there is an excess of uric acid in the blood (uric acidæmia) and it causes severe spasm of vessels in the areas of the hands and feet, we can easily account for all the phenomena of Raynaud's disease. With regard to (2) I have elsewhere suggested ('British Medical Journal,' vol. i, 1890, p. 67) that great excess of uric acid in the blood may, under certain conditions, destroy and break up the red cells and produce hæmoglobinuria and subsequent anæmia, and I have met with some remarkable cases of recurrent anæmia, leucocythæmia, &c., in which, after iron and arsenic had apparently failed, treatment directed to uric acid met with wonderful, and, at first, quite unexpected, success.

In a word, my previous researches led me to believe that both the Raynaud's disease and the hæmoglobinuria in this case were due to excess of uric acid in the blood (uric acidæmia), and this theory affords a most simple explanation of their evident connection.

Now, the patient was evidently low and run down, urea formation was low, acidity was low; a natural result of this was an excess of uric acid in the blood and a plus excretion in the urine which was found. It is recorded that she was dull, irritable, and bad-tempered (the signs of uric acidæmia; see previous references), also that her temper was worse, or that she had a bad headache at the time of an attack. Then, in an attack, the pulse was relatively slow and of high tension, and a trace showed high tension along with some signs of a faltering heart; the heart sounds again (a reduplication of the first sound to the right of the apex, and a loud second sound at the right base) are those commonly met with in the high tension of uric acidæmia. To anyone acquainted with the uric acid headache (migraine) the parallel of symptoms must appear remarkable, and, as in migraine, again she had a pale face and scanty urine during the attack. With regard to scanty urine, I have shown ('Brain,' previous reference, p. 90, and elsewhere) that, as it is in my power to alter arterial tension, so it is in my power to alter the flow of urine; that in the uric acid headache and during the fits of uric acid epilepsy the urine is scanty, but profuse after they pass off, as the uric acid is cleared out of the blood. Now precisely the same thing has been noticed in hæmoglobinuria, namely, that the attack is followed by diuresis (see 'Lancet,' vol. ii, 1889, p. 1086).

I also pointed out in a paper read at the meeting of the British Medical Association at Birmingham in 1890, that the same thing holds for the action of drugs, and I exhibited a diagram from the work of Dr. Lauder Brunton,* showing that when the arterioles are greatly contracted by the action of digitalis or erythrophlenum the excretion of urine is scanty or almost ceases; but when the action of the drugs is coming to an end and the tension falls, there is again a free flow, the exact parallel of what occurs in headache, epilepsy, and hæmoglobinuria.

And lastly, when I treated the uric acid with an acid and a salicylate, the attacks ceased as if by magic, and there was also the most marked improvement in her temper, that is, in the circulation of her brain (see 'Practitioner,' November, 1888, and 'Brain,' Spring Number, 1891, p. 74), so that the result was, to my mind, most certainly *propter* and not *post*.

* 'Pharmacology and Therapeutics,' 3rd ed., p. 430.

It is interesting to remember that in some of these cases where the destruction of blood elements is not sufficiently great to produce hæmoglobinuria it may produce albuminuria; that is to say, we have an albuminuria due to blood conditions and not to kidney lesion, and it is acknowledged by Sir W. Roberts ('Urinary and Renal Diseases,' p. 162) and others that in hæmoglobinuria the blood condition is primary, and any kidney lesion is secondary.

In a paper read before the British Medical Association and published in the Journal (January, 1890) I pointed out that hæmoglobinæmia is probably merely a more advanced condition of the hetero-albuminæmia which, according to Professor Semmola, of Naples, is the cause of albuminuria and of the secondary kidney lesion of Bright's disease, and that, as both hæmoglobinæmia and hetero-albuminæmia may be due to uric acid, we thus get a connecting link between the high-tension pulse of uric acidæmia (see Journal, February, 1889) and the Bright's disease, which according to the late Dr. Mahomed, is its frequent sequel, and the present paper brings out further evidence pointing in the same direction.

I will not prolong this paper by further argument, but those interested in the matter will find a great deal of evidence bearing upon it in a work on the whole subject of the causation of disease by uric acid which is now published (previous reference).

I must not conclude without again expressing my indebtedness to Dr. Wheaton for his careful notes of the time relations of the symptoms and phenomena, and for his examination of the blood, which have added very greatly to the interest of the case.

I see quite clearly that in bringing forward, as I do to-night, the results in a single case I lay myself open to the charge of basing very much upon very small foundations.

I will say at once that my object in bringing it forward is to suggest a treatment rather than to prove a proposition.

I have observed other cases and have applied in this case the treatment suggested by my observations. I have recorded elsewhere ('British Medical Journal,' vol. i, 1890) my argument as to the causation of the disease, and in the above-mentioned work, I have gone into the matter still further.

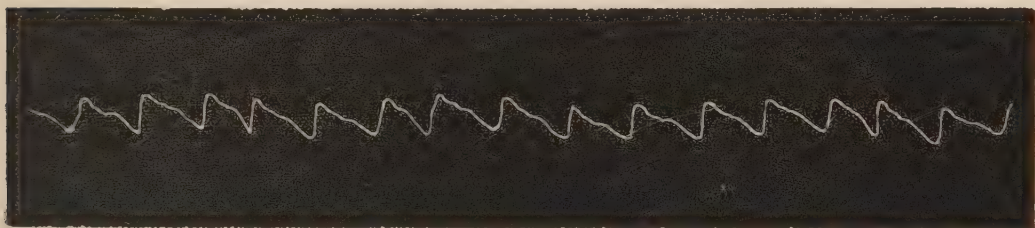
The drugs I made use of in this case certainly appeared to have a very wonderful effect, and, so far as I know, they have not been

used before, in the way I used them, in the treatment of this disease.

If others will make use of them in similar cases we shall soon see whether my result was an effect or a coincidence.

Eliza H —, aged 6.

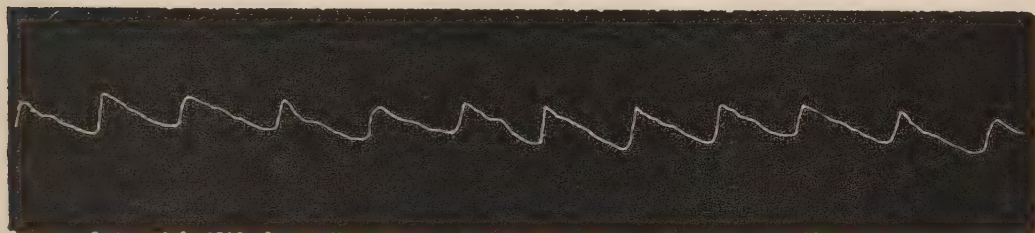
Raynaud's Disease and Paroxysmal Hæmoglobinuria.



Pulse Traces 2nd December, 1890, between Attacks.

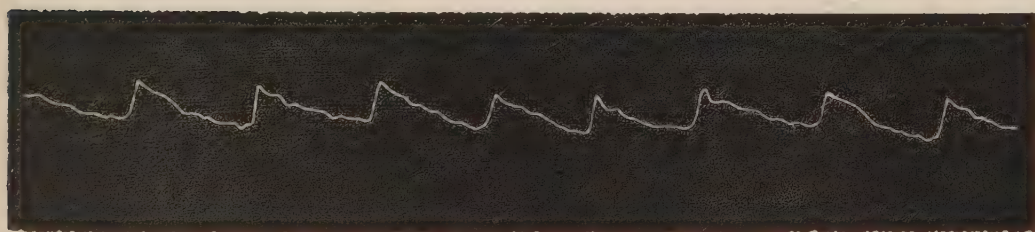
Pulse irregular in force and time. Rate, 105.

Heart, 1st sound reduplicated ; 2nd sound loud.



Trace 12th December, 1890.

Pulse irregular in force and time. Rate, 76.



Trace 24th February, 1891.

Pulse, 80 ; rather lower tension than the others. Is now on salicylates and acids. Temperature much better since these drugs were begun.

Raynaud himself says ('Thesis,' p. 170, French edition) that the great point would be to find a medicament that has a permanent relaxant effect on the smooth muscular fibres of the arterial coats.

For reasons given elsewhere, I believe that the drugs I made use of have such an effect on the arterioles, both in Raynaud's disease and many other conditions associated with contracted arterioles and high tension.

Dr. HUNTER had been working at the subject of blood destruction, the causes of which were extremely obscure. In a number of the diseases of blood of a destructive nature, such as leucocythæmia and pernicious anæmia, the uric acid was greatly increased; there was evidently a direct connection between the two, and he felt sure that the appearance of the uric acid was a result, and was not to be regarded as standing in a causal relation to these diseases. There was no relation between the action of certain substances on blood outside the body and that which took place when the blood was circulating within the vessels. He thought that a more tenable explanation of hæmoglobinuria was that which regarded it as a physical process of neurotic origin, leading first to a local dilatation of vessels, and thus exposing corpuscles, which were perhaps weak in themselves, to a prolonged action of cold, and as a result they died, and hæmoglobinuria followed.

Dr. HAIG said, in reply, that the acid mixture and the salicylate of soda mixture were given on the same day, the acid before meals and the salicylate after meals, each three times a day. With regard to Dr. Hunter's remark that the uric acid was only occasionally in excess in the urine, he said that this point had been gone into at great length in his paper, and he regretted it could not be read in full. He believed it was due to the separation of the urine being very imperfect, the child had an attack lasting half an hour; but the urine obtained would often be that of three or four hours, of which the attack was only one-eighth part. He had had previous similar experience with the urine of headache and epilepsy about which he had written elsewhere. When the urine of the attack could be got separately there was no doubt about the excess of uric acid, and he had found a very large quantity in a more severe case of Raynaud's disease previously under his care. With regard to splenic leucocythæmia, he believed the uric acid was the cause of the anæmia and leucocythæmia, rather than the blood destruction the cause of the excessive uric acid. He had had some interesting experiences with this disease which he intended to publish when his investigations were more advanced. In the experiments he had quoted the creatinin was injected into the blood stream, not merely added to blood outside the body; they were recorded in 'Comptes Rendus de la Soc. de Biologie,' 1877, p. 159. He himself, however, inclined to the view that the action of uric acid in causing destruction of red cells was indirect and due to its influence on the circulation in the liver, the spleen, and the skin (see 'British Medical Journal,' vol. i, 1890, and elsewhere).

January 4th and 18th, and February 1st, 1892.

THE LETTSOMIAN LECTURES: SURGICAL TREATMENT OF TRIGEMINAL NEURALGIA.

By Professor WILLIAM ROSE, M.B., B.S. Lond., F.R.C.S.

LECTURE I.

Introduction.—Ætiological Classification.—Intracranial, Cranial, and Extracranial Causes, &c.—Pathological Anatomy.—“Epileptiform Neuralgia.”—Clinical Picture.—Differential Diagnosis.—Surgical Treatment discussed generally:—Neurotomy—Neurectomy—Nerve-extraction—Nerve-stretching—Ligature of Carotid.

MR. PRESIDENT AND GENTLEMEN,—On occasions like the present it is the pleasurable duty of the Lettsomian Lecturer to commence by thanking you and the Fellows of the Society for the honour conferred upon him, and although my acknowledgment be brief, I am in no way behind my illustrious predecessors in my appreciation of this distinction.

The difficulty of selecting a subject which shall touch on new ground increases as years roll by, and in considering what I could best bring before your notice it seemed to me that an account of my recent experiences in the treatment of intractable trigeminal neuralgia would be interesting to the profession generally, and acceptable to you. And the more do I feel this, since on glancing over the list of subjects dealt with by the distinguished men who have preceded me in this position, I observe that with the exception of the year 1866, when the late Dr. Anstie delivered a course of lectures on “Some Painful Affections of the 5th Nerve,” this subject has not been touched upon.

The fact that up to the present time this disease has baffled all the attempts of medical and surgical treatment marks it out as one that demands further attention and the freest discussion both from the pathological and clinical standpoints. Details as to the morbid anatomy of this disease are extremely scanty; indeed it has by no means received the attention from pathologists that it deserves, although recently two or three important papers have

appeared in the American journals dealing with the pathological conditions of the nerves and blood vessels, to which I shall allude later. In future, wherever practicable, the portions of resected nerves should be examined microscopically, and every opportunity taken of learning more of the local conditions which may be present; all cases operated on should be kept under observation, if possible, for years, so that reliable statistics may ultimately be obtained. The great majority of recorded cases are diminished in value from the fact that this precaution has not been adopted. The plan I propose to follow in these lectures is to give a review of the various surgical methods adopted in the treatment of this disease after a short introduction, devoted to its causation, pathology, and symptoms, inasmuch as an accurate estimate of these must be formed as a basis of scientific treatment; and I believe I shall be able to demonstrate, both by the experience of others and my own, that surgical treatment of the trunks of the 5th nerve in the severest forms of this affection has not been attended with anything like average success, and to submit a more radical means of dealing with this complaint in the extirpation of the Gasserian ganglion, providing that this can be accomplished without risk to life, and with, let us hope, every prospect of permanent relief.

The frequency of this particular form of trigeminal neuralgia is such as to constantly obtrude itself before the general practitioner, and its distressing character warrants even the most severe measures being taken for its treatment. From the statistics of the Bonn clinic given by Conrads in 1889, it is observed that out of 717 cases of neuralgia under treatment, 239 were of the 5th nerve, a number only slightly exceeded in the sciatic, in which 243 cases were met with, whilst only 112 instances of intercostal neuralgia were present, and 54 of cervico-brachial.

With regard to the causes of this disease, I would suggest the following as a convenient *ætiological classification* :—

- I. Intracranial—cerebral;
radical;
ganglionic.
- II. Cranial.
- III. Extracranial or peripheral.
- IV. Toxic, *e.g.*, mercury, lead, or malaria.
- V. Reflex.
- VI. Functional.

Amongst the *cerebral* causes may be mentioned sclerosis, and aneurisms or tumours interfering with the deep origins of the nerve in the pons and medulla. Probably some of the toxic bodies may act upon the centres and thus produce neuralgic pain, whilst syphilis is a potent factor in its production, if we may argue from analogous conditions of the nerve centres elsewhere.

Dr. Putnam*, in an able paper, has recently advanced some important points for consideration in reference to the central causation of neuralgia, and the effect of central disturbances upon the peripheral nerve trunks, along which the morbid nervous stimuli pass. He suggests that the following predisposing factors of trigeminal neuralgia exist, viz. :—

(i.) The large extent of the deep origins, and hence the existence of wide connections with other nerve tracks rendering this nerve more liable to be affected at its origin by morbid influences from many extraneous sources independent of its own area of distribution ; and

(ii.) The elaborate and delicate organisation of the centres as evidenced by the exquisite sensibility of the parts supplied by it, *e.g.*, the lips, tongue, and eyes.

He also maintains that a peripheral neuritis may be the sequence of severe and frequent nerve storms passing along a particular nerve track, emanating from a diseased centre, basing his argument on the fact that even physiological action produces demonstrable changes in the shape and size of a cell-nucleus.

Radical causes.—The sensory root of the 5th nerve between the exit from the pons and the Gasserian ganglion may also possibly be affected. The dural sheath and the aperture in the dura mater through which the nerve passes may from inflammatory thickening become an important item in originating the trouble. In one of my cases of removal of the Gasserian ganglion this opening in the dura mater was seen, and it certainly appeared small in comparison with the size of the nerve passing through it.

Ganglionic causes.—The Gasserian ganglion itself may be the seat of chronic inflammatory or interstitial disease, leading to sclerosis and subsequent pressure on the nerve cells. The first case in which I removed the ganglion, an account of which is

* 'Boston Medical and Surgical Journal,' August 13th and 20th, 1891.

given in the 'Medical Society's Transactions' (vol. xiv), with a woodcut of the microscopic section, is presumably an instance of this (Fig. 1). It is interesting to compare the statement of

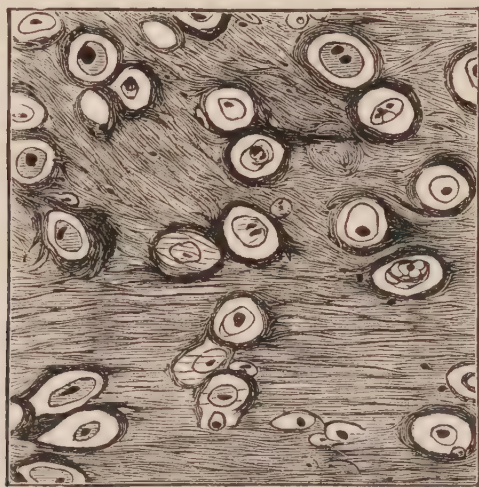


FIG. 1.—Section of a portion of the Gasserian ganglion in Case I, showing thickened and sclerosed connective tissue, and irregular shape of ganglion cells. Seen under 1-5th inch objective.

Carnochan as to the importance he assigned to Meckel's ganglion in infra-orbital neuralgia. He says:—"I believe that in such aggravated cases of neuralgia the key of the operation is in *the removal of Meckel's ganglion or its isolation from the encephalon*. Where even a large portion of the second branch of the 5th pair has been simply exsected from the infra-orbital canal, the ganglion of Meckel continues to provide to a great extent the nervous ramifications which still maintain and keep up the diversified neuralgic pains. Besides, the ganglion of Meckel, being composed of *gray matter, must play an important part as a generator of nervous power*, of which, like a galvanic battery, it affords a continual supply; while the branches of the ganglion, under the influence of the diseased trunk, serve as conductors of the accumulated morbid nervous sensibility."*

As to the *Cranial* causes, the fact that all the branches of this nerve have to pass through bony canals on their way to their peripheral distribution is another element which must not be lost sight of in considering the causation of this disease. In the first place, in fractures of the base of the skull, implicating any of the foramina, subsequent formations of new bone in the process of

* 'Amer. Jour. of Med. Sci.,' 1858, p. 136.

repair may press injuriously upon one or other of the trunks; and we can also imagine that a syphilitic periostitis may diminish their lumen with a similar result. Bony or malignant tumours connected with the base of the skull, and extravasations of blood, from meningeal hæmorrhage, for example, might induce pressure, either immediately or subsequently from cicatricial development. Mr. Carless has made a number of careful observations on a series of skulls in the museums of King's College and the Royal College of Surgeons, as to the size and relative position of the foramen ovale, and the results he arrives at are very interesting. The shape and size of the foramen ovale, he states, are not constant, and differ in every conceivable way. Thus not only does the form vary from almost a circle to that of a long narrow slit, but the absolute calibre is by no means constant, and the foramina on the two sides of the skull by no means symmetrical. As a rule, however, the foramen is rightly named "oval," the long diameter being inclined backward and outward. The length varied from 6·5 to 9·5 mm. in six male skulls measured, the average being about 7·6 mm.; whilst in twelve female skulls examined the variations were from 5 to 8 mm., the average being 7 mm. on the right side and 6·5 on the left, *i.e.*, the long diameters of the foramina were distinctly less in female skulls. In breadth, the foramina in the male skulls varied from 3 to 6·5 mm., averaging 4·7 mm. on the right side and 4·2 mm. on the left. In the female skulls the breadth varied from 2 to 5·5 mm., the mean being 3·4 mm. on the right side and 3·7 mm. on the left, an interesting point to note in connection with the fact that all the patients from whom I have removed the Gasserian ganglion have been females, and the disease has existed in all on the right side.

The varieties of shape which the foramen assumes can be gauged by the following measurements of some extreme cases:—In one old massive male skull the foramina on each side measured $9\cdot5 \times 4\cdot5$ mm., whilst in an edentulous female skull the measurements were $8 \times 2\cdot5$ mm. and 7×2 mm., on the right and left sides respectively; and still in another, $7\cdot5 \times 6\cdot6$ mm., the foramen being nearly circular. The average size of the foramen in a male is about $7\cdot5 \times 4\cdot5$ mm., and in a female $6\cdot7 \times 3\cdot5$ mm. In one skull examined the foramen was very large and quite heart-shaped. The disproportion often existing between the foramina on the two sides of the skull will be evidenced by the fact that of

the eighteen measured in only one case did they actually correspond, although in three others the measurements were nearly alike (Fig. 2).

Average in male 7.5×4.5 mm.



Average in female 6.7×3.5 mm.



Old male skull 9.5×4.5 mm.



Edentulous female skull.
R. side 8×2.5 mm.



L. side 7×2 mm.



Old female skull 7.5×6.6 mm.



FIG. 2.—Varieties in shape and size of foramen ovale (*actual size*).

The size of the foramina does not seem to change at all regularly with the age, for whilst some edentulous skulls have large foramina, others have small; and young skulls show similar variations. The largest foramina occur in heavy thickened skulls, and the shape and size of the cranium seem to influence that of the foramen much more than does either the age or the sex. Perhaps it may be correct to say that *relatively* the foramina are larger in younger skulls, and in those of the male sex.

Under *extracranial* or *peripheral* causes we include any definite peripheral lesion affecting the nerve trunks from their point of exit from the skull to their ultimate fibrillæ. Implication of the nerve in cicatrices, impaction of foreign bodies, pressure from tumours, all find their place here, as well as many other conditions; but special attention must be devoted to two or three of these, *e.g.*:—

1. Those connected with the teeth.

It goes without saying that dental caries is a fertile cause of

neuralgia, and that often of a most intense character. Neucourt and Friedberg have rightly emphasised it, noting the fact that the tooth which is the real cause of the trouble is not necessarily painful. They also declare that the pain may sometimes be cured by extracting a non-painful carious tooth, whilst on the other hand it may occasionally be increased by extracting a painful one. In some cases it may appear to start from a sound and painless tooth upon the same side.

Gross states that the facial neuralgia may be caused by the alveoli of edentulous jaws becoming filled with bone, and so compressing the nerve filaments; he has relieved the pain in these cases by either removing small wedges of bone or by drilling into the affected area.

2. The condition of the peripheral bony canals through which the nerves pass, *e.g.*, the infra-orbital and inferior dental, is another most important matter for consideration. The space within is very small, and any periosteal swelling or deposit of new bone would diminish the lumen, and thus compress the nerve. In more than one instance I have found this condition in the inferior dental canal; unfortunately when once neuritis has been started by pressure in this way, it often has a tendency to spread. Thus, in the first case in which I removed the ganglion, the original cause was evidently, as shown by a former operation, pressure upon the dental nerve by a new formation of bone. This was temporarily relieved by the earlier operation of trephining the jaw, but recurred, needing more severe measures. It is a significant fact that in children, when relatively the bony canals are widest, severe neuralgia is practically unknown.

3. Exposure to damp or cold may be legitimately looked upon as an exciting case of peripheral neuralgia by setting up a rheumatic perineuritis, causing exudation into the nerve sheath and pressure upon the *nervi nervorum* leading to permanent organic change.

Retention of secretion in the frontal sinuses has been known to cause neuralgia (Horner and Seelig Müller); as also diseases of the ear (Tröltzsch and Moss).

The last three divisions in our ætiological table, *viz.*, the *toxic*, *reflex*, and *functional* groups, will receive but slight notice here. At the same time I cannot help being impressed with the fact that in a large majority of neuralgic patients there is a distinct neurotic

temperament, inherited from the parents or acquired, which undoubtedly forms an element amongst the predisposing causes of this disease. Indeed, Dr. Buzzard* goes so far as to say that, in many cases of epileptiform neuralgia, especially of the third division, there is an hereditary tendency to insanity. A marked condition of anæmia is constantly exhibited by many of these patients, and was illustrated by my illustrious predecessor, Dr. Anstie, in his famous dictum "Neuralgia is the prayer of the nerve for blood." But although these functional defects play an important rôle in the determination of the neuralgic stage, they still can be alleviated, and should not be allowed to continue, otherwise, as has been already pointed out, the functional trouble may merge into an organic lesion of a permanent and progressive character, which can only be relieved by very serious operative measures. Mental anxiety and worry are often looked upon as being closely connected with the production of neuralgia, but we are in ignorance as to the manner in which this is brought about.

A marked distinction should be drawn between those cases, on the one hand, of simple neuralgia dependent on slight functional derangement, which can generally be cured by medicinal treatment, and those severe and intractable forms known as *tic douloureux*, or Fothergillian neuralgia, which was also christened "*tic epileptiforme*," by Trousseau, and to which more recently the name "*Epileptiform Neuralgia*" has been applied. This latter name is, perhaps, a more suitable one than would at first appear; for although the motor nerves are but slightly and secondarily affected, yet the explosions of nerve force, or convulsions of pain, along the sensory nerve tracks bear a certain resemblance to the typical motor phenomena ordinarily associated with the term epilepsy.

This severe variety is more common in the second and third divisions of the nerve than in the first, the infra-orbital and inferior dental branches being especially liable to it. The simple form would appear to more commonly affect the ophthalmic division, and it is now generally acknowledged that supra-orbital neuralgia is more closely associated with central neurosal conditions, such as migraine, &c., rather than with *tic*. The amenability of this form to quinine, and the frequent periodicity of the attacks has suggested the term "*browague*," so often applied

* 'Quain's Dictionary of Medicine,' see article on "*Tic*."

to it, although there is no absolute evidence connecting it with malarial poisoning.

An interesting paper was contributed at the beginning of last year by Professor C. L. Dana* to the American Society of Neurology, dealing with the pathology of this disease, and based on the microscopic examination of the excised nerves in several cases. He admits that many inveterate trigeminal neuralgias are due to local lesions, such as exostoses, aneurisms, syphilitic growths, &c.; but maintains that the cases of bad tic commonly seen commencing in the superior maxillary branch or in the inferior dental after middle life are of an entirely different nature. He does not think that the pain is due to neuritis or nerve degeneration, because, as a rule, there is no permanent anæsthesia; whilst in cases of progressive trigeminal anæsthesia from a degenerative neuritis there is, as a rule, but little pain. The former of these propositions can hardly hold good, inasmuch as even when the nerve has been removed very slight anæsthesia remains permanently, other communicating nerves apparently taking on the duties of the one removed. In most of the nerves examined by Professor Dana no degenerative change could be found in any of the nervous tissue proper, but very marked and noteworthy pathological conditions were observed in the arteries accompanying the nerves; and he considers that many such cases are due to an obliterating endarteritis of the nutrient vessels of the nerves (Fig. 3). The reasons he adduces for this opinion are the following:—

1. The disease occurs at that period of life when arterial changes begin.

2. It affects chiefly and primarily a nerve supplied by one of the branches of the internal maxillary artery, *i.e.*, either the infra-orbital or inferior dental. As has been already pointed out, the supra-orbital nerve is but rarely the seat of epileptiform neuralgia, and it is a noteworthy fact that it is supplied by a branch from an entirely different arterial system, *viz.*, the internal carotid. Hence the disease follows a certain fixed vascular distribution.

3. Microscopic examination of excised nerves demonstrated a striking arterial degeneration in three out of five specimens examined; in the other two nerves no arterial twig was visible in the sections.

* 'Journal of Nervous and Mental Disease,' No. 1, 1891.

4. Therapeutic experience strengthens this view in that nitroglycerine will occasionally give relief quickly, the effect lasting for some time. Aconite, too, by diminishing the pulse tension, is used with advantage.



FIG. 3.—Section of arteriole in a condition of obliterative endarteritis from nerve excised for epileptiform neuralgia. (After *Dana*.)

5. As there is no doubt that removal of the peripheral nerves sometimes cures tic entirely, he argues that the disease must be peripheral and due to some local peripheral cause.

6. MM. Quenan and Lepars have recently stated that by a new means of injection they have discovered a closer and more extensive relationship between the vessels and the nerves than was hitherto suspected, and suggest that disturbances in blood supply may be an element in producing neuralgia.

Professor Dana admits that there are weak points in this theory,

and only claims that the arterial change is one of the factors in the production of the severe cases of tic, whilst a special vulnerability of the central nervous system is another essential predisposing condition. Time and further observation will alone decide whether Professor Dana's theory is correct in giving a causative position to this arterial change.

Independently of this paper, Dr. Putnam* described the same process as evident in some of the nerves that he had examined. He states that in one or two cases the tunica intima of the blood vessels was greatly thickened and the lumen of the vessel encroached upon and even obliterated by a dense tissue containing fibres and nuclei. In one or more large vessels affected in this way, the central mass of tissue appeared to be attached to one portion of the vessel wall, and opposite this the fenestrated membrane, which everywhere else was perfectly distinct, had entirely disappeared (Fig. 4).



FIG. 4.—Section of nerve showing obliteration of small arteriole in a case of epileptiform tic by a mass of fibro-cicatricial tissue growing in from the wall at a spot where Henle's elastic membrane is absent.

A. Tunica adventitia.

B. Tunica media.

C. Tunica intima and Henle's membrane. (After Putnam.)

I cannot add much from personal observation to this interesting idea, but certainly in several of my cases I have been much struck

* Putnam, 'Boston Medical and Surgical Journal,' Aug. 20th, 1891.

with the size of the vessels and the substantial thickness of their walls; and this was particularly the case in the inferior dental artery of a patient on whom I recently operated; moreover, in one of the microscopic specimens made for me by Mr. Turner, of the tissues removed in the third case in which I endeavoured to extirpate the Gasserian ganglion, this condition appears to be present in what is probably a portion of the small meningeal artery.

The majority of observers do not agree with the statement of Professor Dana that there are no definite changes in the nervous elements. Horsley,* Schweinitz,† and Putnam‡ all speak very definitely of microscopical appearances of a sclerosing nature, and this I can fully confirm. The axis cylinders are found swollen or shrunken, and sometimes have disappeared entirely; they are usually difficult to stain with either logwood or carmine. The medullary substance often appears swollen, and the sections of the tubules are not clear and sharply defined, but consist of a confused mass of concentric rings. "The transverse section of nerve fibres might in places be not incorrectly compared to the interior aspect of a minute oyster-shell both in shape and by virtue of this peculiar concentric arrangement" (Schweinitz). The sheath of Schwann is crumpled up more or less, and its nuclei increased in numbers. The interstitial connective tissue (or endoneurium) is also increased in amount, especially around the blood vessels. These appearances are similar to those seen in a case of chronic neuritis, and such must be of an ascending character, as the changes are often more marked peripherally than at the central end. Putnam notes the fact that all the fibres in the nerve are not invariably affected in the same way, and often healthy and diseased tubes may be seen in the same section lying side by side. The perineurium is also in some cases thickened, and some observers have noticed small nerve bundles lying in this tissue with the fibres very much changed. These are probably the remains of the "nervi nervorum" described by the late Mr. John Marshall, the compression of which may possibly be one of the chief factors in the production of the pain (Fig. 5).

* Horsley, 'Transactions Odontological Society,' 1887, vol. xix, p. 270.

† Schweinitz, v. paper by Ewing Mears, 'Transactions American Surgical Association,' 1884, vol. ii, p. 469.

‡ Putnam, *op. cit.*

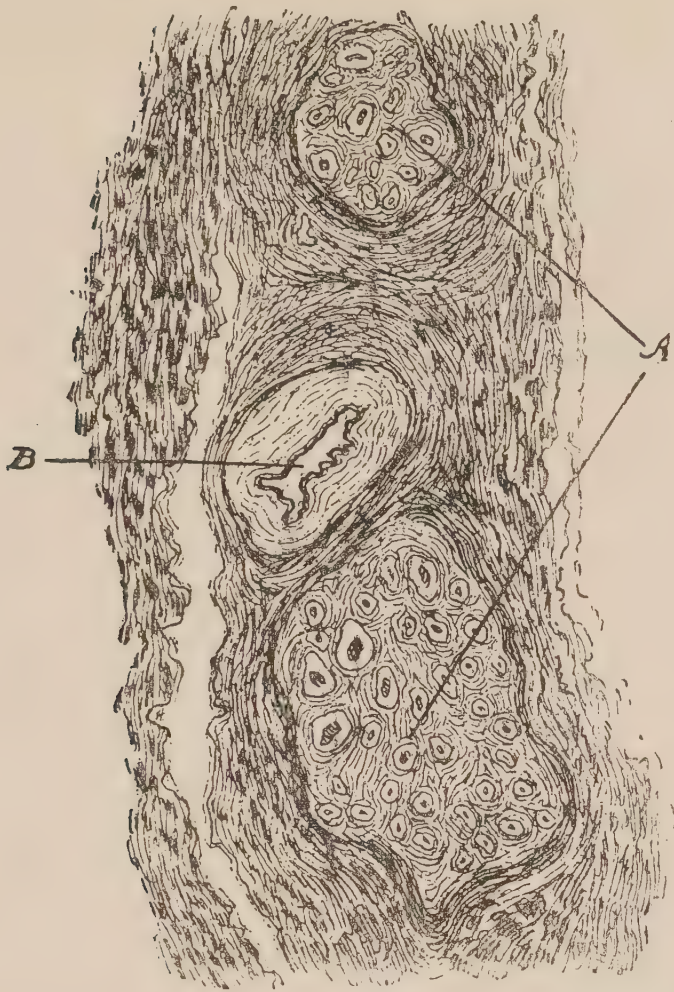


FIG. 5.—Section of nerve in a case of epileptiform tic, showing the irregular condition of the nerve fibrillæ, and particularly of the sheath of Schwann (A) ; there is some increase of the connective tissue. An arteriole (B) with thickened wall is also seen. (After *Putnam*.)

Leaving the pathological question, we will now turn to the clinical aspect, to discuss the leading symptoms which are usually met with in a typical case.

The patient, usually a female and probably in the middle period of life, complains of more or less periodic attacks of severe unilateral cumulative pain, darting along special nerve tracks, and referred particularly to the points of exit of the peripheral branches upon the face, and especially to those of the second and third divisions. The attacks, after reaching a certain pitch of severity, suddenly subside, leaving the patient intervals of freedom, which, as the complaint advances, become proportionally diminished. As these intervals become shortened, so does the general tone of the nervous system become sympathetically lowered from anxious

apprehension and dread of the return of the pain. As a severe paroxysm of pain frequently follows any touching of the affected parts, patients guard them most jealously; washing even is avoided; and I have seen many such cases where the side of the face was distinctly dirty from this cause. Similarly the tongue may become thickly covered with fur on the affected side in consequence partly of the patient using only the opposite side of the mouth in mastication, and possibly also to trophic disturbance.

When once the hyper-excitability of the nerve centres has been established, so that stimuli from without cannot be retained, but, as it were, overflow tumultuously into definite painful tracks, it matters not where the stimuli come from, the same result always obtains. Thus the patient gets into a morbid state of dread, since any unexpected event, such as the mere slamming of a door, a sudden noise, or a draught of air, is sufficient to precipitate an attack. In the midst of speaking, laughing, coughing, and particularly chewing, a paroxysm occurs with lightning-like rapidity, causing a sudden cessation of any of these acts. The paroxysm dominates the unfortunate patients completely for a time; they frequently hold their hands to the face evincing unmistakable signs of intense suffering. Cases are on record, however, where a sudden cure has resulted from the reception of some unexpected or startling shock, whether physical or psychological, *e.g.*, Sir Henry Lawrence's well-known case, in which he was cured, at least for a time, of neuralgia by the receipt of the news of the Indian Mutiny calling him to instant action.

The special character of the pain must be carefully noted, being entirely different to those migratory or wandering pains which often attack these patients in the intervals between the paroxysms of true tic, and which might mislead the medical attendant in arriving at a decision as to the exact seat of the lesion. Those who have long suffered from this disease look upon them as trifling in comparison with the agony of which they are so frequently the precursors.

But, in addition to the preceding symptoms, a typically severe paroxysm includes other phenomena or complications referable to the motor, vasomotor, and secretory apparatus, together with disturbances of the special senses.

The effect upon the facial muscles through the intimate connections which exist between the terminal filaments of the 5th and

7th nerves is to induce, firstly, spasmodic contraction as evinced by contortions of the face, and later on paralytic symptoms. The muscles of the eyeball may become similarly affected, and ptosis and external strabismus have been observed from paralysis of the motor oculi nerve. During a convulsive seizure the muscles of the jaw supplied by the motor fibres of the trigeminal, as also those supplied by the facial, often become the seat of clonic spasm, which may also extend to those rotating the head.

The vasomotor disturbances most commonly met with are unilateral hyperæmia of the affected side, suffusion of the conjunctiva, and in cases of long-standing disease tumefaction from venous congestion and consequent inflammatory hyperplasia in the lips, cheeks, sides of the tongue, and floor of the mouth. Partly due to this vasomotor disturbance, and partly to irritation of the excito-secretory fibres is the unilateral sweating of the forehead and side of the face so frequently observed. From a similar cause excessive lachrymation occurs during an attack combined with an abundant flow of nasal mucus and of saliva.

The temporary and permanent effects upon the special senses demand a passing notice. Impairment of vision is present in a small percentage of cases, and is probably due to a trophic disturbance in the retina itself, but when temporary may also arise from a transient paresis of the ocular muscles. The sense of hearing is but rarely affected, whilst taste and smell are only in exceptional circumstances interfered with. Even in severe neuralgia of the lingual nerve with intense hyperæsthesia of the affected side of the tongue, the sense of taste may be quite normal.

Another manifestation of this disease which was formerly looked upon as one of considerable diagnostic importance is that of localised areas of hyperæsthesia, the so-called *points douloureux* of Valleix. They are usually located at the points of exit of the terminal branches of the nerves (Fig. 6), but occasionally secondary foci are found in other situations. Thus, with neuralgia of the *ophthalmic* division, there is often a tender spot on the scalp a little above the parietal eminence, rendering brushing the hair painful and even impossible. The more common sites of hyperæsthesia are the supra-orbital notch, the upper eyelid, the eyeball, and the junction of the nasal bones and cartilage. As I have previously stated, the neuralgic affections of this division are not so

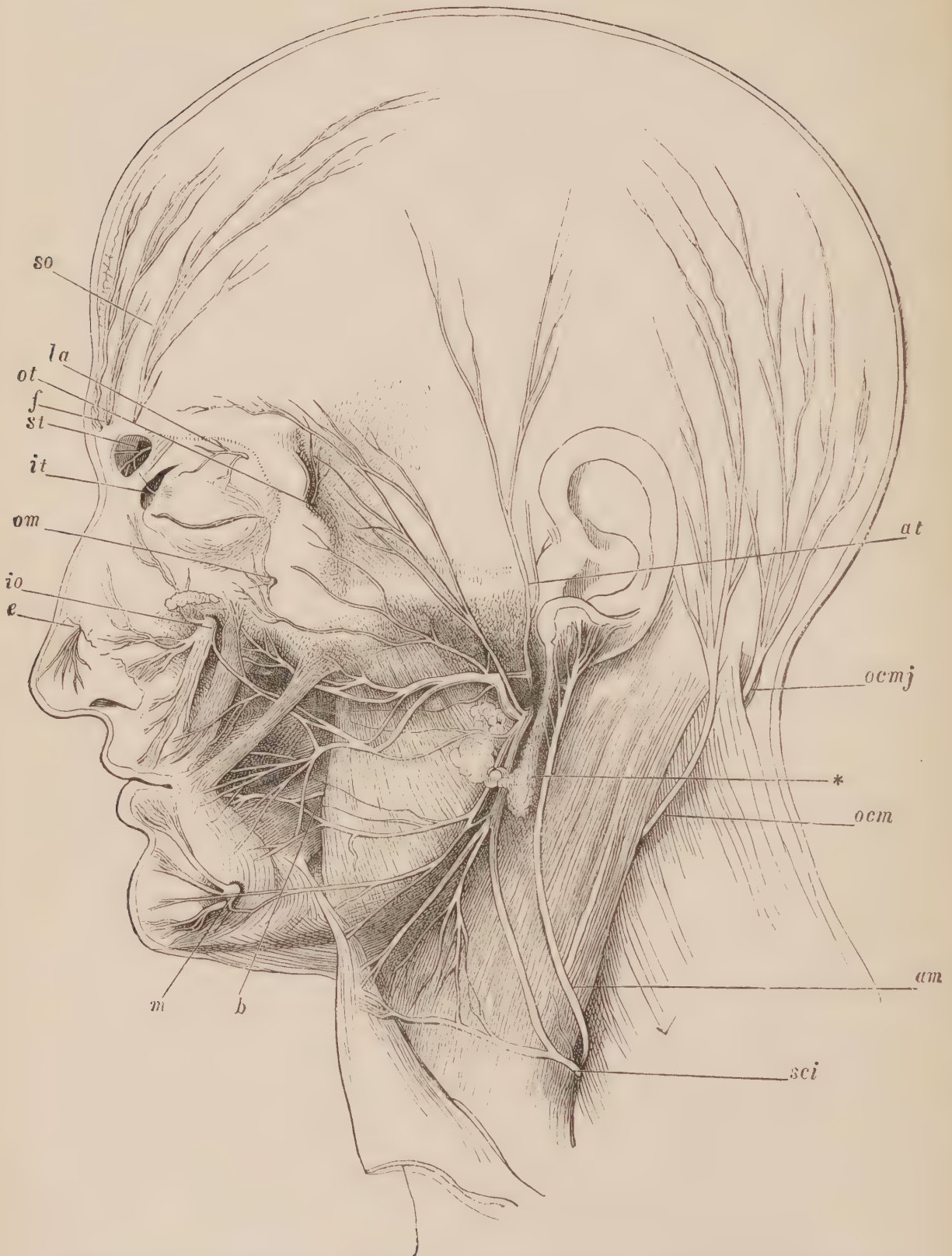


FIG. 6.—Area of distribution of 5th nerve. Terminal branches of the facial and sensory nerves of the face; the parotid gland* is almost completely removed. (MacCormac.)

so. Supra-orbital.
la. Lacrymal.
ot. Temporal.
f. Frontal.
st. Supra-trochlear.
 Infra-trochlear.

om. Malar.
io. Infra-orbital.
e. Nasal branch of ophthalmic.
m. Mental.
b. Buccal.

sci. Superficial cervical.
am. Great auricular.
ocm. Small occipital.
ocmj. Great occipital.
at. Auriculo-temporal.

severe, and seldom assume the true characters of the epileptiform variety. In the *second* division these *points douloureux* are found at the infra-orbital foramen, over the malar eminence where the temporo-malar branch emerges from the bone, at some spot along the gum of the upper jaw, and even in the palate at the orifice of the posterior palatine canal. As to the *third* division, the common situations of these tender spots are at the mental foramen, the side or tip of the tongue, the lower lip, and between the angle of the jaw and the ear. It is more than doubtful whether we can place any diagnostic value upon these tender spots, inasmuch as they are not usually manifest in the earlier stages of the disease; and when at all pronounced they probably indicate a localised organic change of the tissues in the immediate neighbourhood.

The trophic influence of the nerve over the parts supplied by it is shown in the nutritive changes met with in some instances. The skin tends to become thin, shiny, and atrophic, similar to the condition known as the "glossy skin," described by Sir James Paget many years ago. This may be partly due, however, to the violent friction of the parts and the application of counter-irritants used in the treatment, but cannot be wholly accounted for in this way. Herpetic eruptions along some of the peripheral branches are also occasionally seen.

DIFFERENTIAL DIAGNOSIS.

In dealing with any case it is important to ascertain, if possible, the exact site of the lesion. The previous history and surroundings of the patient should be carefully investigated, special stress being laid on possible injuries to the soft and hard structures in any part of the area of distribution of the nerve. The teeth should be examined by a skilled dentist, and old stumps removed. In women the condition of the uterus and ovaries and the regularity or not of the menstrual functions should also be noted.

In spite, however, of the greatest care, it is often difficult to differentiate between central and peripheral neuralgiæ. Benedikt maintains that in central cases the pain is more lancinating, more widely diffused, localised in the bones and not limited to any particular nerve tracks, whereas in the peripheral variety the opposite conditions are present; this, however, cannot be relied upon from the fact that a local lesion may morbidly react upon the

nerve centres, and so make diagnosis impossible. Billroth suggested, as a test, the employment of hypodermic injections of morphia, maintaining that if a tic persists in spite of three grains of morphia taken internally, or a quarter of a grain injected into the cheek, the neuralgia is certainly of peripheral origin.

If it be conceded that operative treatment in central neuralgia is useless, then the importance of ascertaining the locality of the lesion becomes manifest. I am not, however, prepared to say that in cases of central origin operative interference is of necessity unavailing, inasmuch as we cannot tell what reactionary influence of a beneficial character may follow the removal of the peripheral branches, especially when this is carried out with thoroughness and as near to the brain as possible.

Two theories have been adduced to explain the advantageous effect of operative treatment in these central cases. The first of these was propounded in 1830 by Bell, and has since received the weighty support of that distinguished neurologist Erb; they maintained that the peripheral activity excited by the operation acts as a counter-irritant to the centres, and by its alterative and tonic effects assists in restoring them to a healthy state. If such be true, then severe and radical operations are entirely needless, and should be avoided; a limited operation, such as nerve-stretching, which can be repeated as often as necessary, would be sufficient. The test of experience, however, has not confirmed this hypothesis, inasmuch as the repeated recurrences of pain have compelled surgeons to undertake more and more radical and heroic measures. The second theory, and that which seems more in conformity with the ideas and practice of the present day, is that of Wagner, who maintained that the removal of the peripheral nerves presumably affected places the centres in a position of rest by excluding the afferent stimuli, and thus assisting, for a time at least, healthy repair in the centres. The logical deduction to be drawn from this doctrine is that the more extensive and radical the operations, the more likely are they to be attended with success.

TREATMENT.

It is not my intention to deal with the *medical* treatment of neuralgia, beyond a passing allusion to the more prominent of the drugs which have held for a time a reputation for alleviating or

curing this complaint. Amongst these may be noted quinine, aconite, cocaine, chloral hydrate, croton-chloral, antipyrin, arsenic, paraldehyde, phenacetin, gelsemium, and the alkaline bromides and iodides. Locally aconite, belladonna, and menthol are commonly used, whilst blisters are also frequently employed. Many of these have undoubtedly proved beneficial; but probably few cases have been finally cured, when once the neuralgic habit has been fully established, except perhaps those owing their origin to syphilis, or some obvious and removeable cause. In all probability the cases which find their way to the surgeon are those in which medical treatment, both local and internal, has failed to relieve, and such patients have usually borne with their sufferings for many years before they seek surgical interference.

Hypnotism has of late years been much vaunted and had recourse to by French physicians as a curative measure, and a certain amount of success would appear to have followed its employment. Putting aside the ethical question as to the right of the physician to use such a remedy, it is difficult to understand how, if the neuralgia be due to organic lesion, the occurrence of pain can be prevented by a suggestion given during the hypnotic state.

In reviewing generally the *operative treatment* of this disease, the first and simplest method employed, that of *Neurotomy*, although alluded to by Albinus and Galen, was first carried out on a branch of the 5th nerve, about 150 years ago, by Schlichting and Maréchal. It was performed more or less frequently up to 1840, and then for a decade or more seems to have fallen into disuse, if the silence of records is to be so interpreted. But in 1852 M. Roux, of Paris, recorded four cases of operation on the 5th nerve, since which time they have become numerous enough. The plan usually adopted was that of introducing subcutaneously or under the mucous membrane a fine tenotome, and dividing the nerve, whilst in a few cases the open method was employed. Transient relief followed in some instances, but not in all; and usually the neuralgic pain recurred at an early date with all its old severity. The cause of this is not far to seek, in that we now know how very readily divided nerves reunite, and, even when a portion of a nerve trunk is entirely removed, regeneration of the destroyed portion is a matter of frequent experience. The scar itself, moreover, may become the exciting cause of the relapse, and may require extirpation. To prevent this reunion of the

nerve, *neurectomy* on a more extensive scale naturally suggested itself, and in this country it was performed by Abernethy in 1793. Even then insufficient portions of the trunks were removed, seldom more than half an inch, so that the regenerative energy of the nerve soon bridged over the interval, and the pain recurred. Many other plans were suggested to obviate this. Thus in 1822, Klein crushed and cauterised the central end; Middeldorpff, Bardeleben, and Luikart divided the nerve with the galvano-cautery; Boyer cauterised the peripheral end; whilst Malgaigne split one or both ends longitudinally, and turned them back in a loop, in other cases attempting to place a flap of soft tissue between the two ends. M. Péan, more recently, has recommended the old plan of removing the central end with cauterisation.

The results of all these earlier operations, whether neurotomy or neurectomy, were not satisfactory, as the following statistics will show:—

Otto Weber* collected 100 cases with only eighteen cures.

Wagner, in 1869, recorded 135 cases (possibly including Weber's), and the results given were as under:—

Cures after 3 years, 18.

Fatalities from sepsis, 6.

In 1 case, relief for less than a month.

In 32 cases, relief for months.

In 20 cases, relief for 1—3 years.

In 24 cases, relief temporary, but the after-history was not known.

Dr. Fowler, of Brooklyn,† quotes eighty-three cases recorded since Wagner's list was published; but the statistics are of comparatively little value, as many of them were kept under observation for but a short time.

The regenerative energy of the nerve was shown, however, not to be the only cause of the relapses. Attention was directed to the important fact that so many of the branches of the nerve pass through bony canals, and were thus more directly exposed to pressure; whilst it was also noted that during the period of life, when these bony canals are relatively widest, *i.e.*, among children, neuralgia is practically unknown. The suggestion naturally fol-

* Pitha and Billroth's 'Handbuch,' Bd. iii, Ab. i, Lief. i, p. 163.

† 'Annals of Surgery' (St. Louis), 1886, ii, p. 269.

lowed that the nerves should always be divided on the proximal side of the canals.

As to the extent of the operation of neurectomy, it is eminently desirable to divide the nerve as high as possible, in order to get above any undiscovered lesion, whilst it is also advisable to remove as much of the nerve peripherally as is practicable in the affected locality in order the more certainly to place the nerve centres at rest by preventing stimuli from reaching the brain along anastomosing trunks. For example, the inferior dental nerve should be divided, not only at its entrance into the inferior dental canal, but also at its exit from the foramen ovale, and the intervening portion excised. The removal of a large extent of the nerve trunk was first suggested, I believe, by Professor Braun, as a means of dealing with the infra-orbital nerve; he advised its division at the foramen rotundum, and also at the infra-orbital aperture, removing the whole of the intervening portion by traction. M. Beau, of Toulon, recommended a similar process to be adopted in the treatment of the inferior dental branch, dividing it at its entrance to the dental canal and at its exit from the mental foramen, removing the part between. But it was Professor Thiersch* who carried out this plan of *nerve-extraction* more thoroughly, and extended the operation to all the divisions of the trigeminal.

He frees the nerve from its connections at some spot easily accessible, and then, by a process of torsion with specially constructed forceps, is able to draw out a considerable length of it, both distally and proximally, tearing the central end away, and cutting the peripheral. By this means very large portions of the nerve can be removed, and so, it is hoped, recurrence of the neuralgia more effectually prevented. He recorded twenty-eight cases of the operation in 1889 performed on seventeen different patients, having operated eleven times on the infra-orbital, five times on the supra-orbital, four times on the inferior dental, thrice on the lingual, and once on the mental. Five or six centimetres can be removed from the supra-orbital; and he showed two specimens of the lingual nerve that had been extracted from the cadaver 9.5 and 15 cm. in length respectively. Of the second specimen, 6 cm. were central, and 9 cm. peripheral, measured

* 'Deutsche Gesell. f. Chirurg.,' 1889, p. 44.

from the angle of the jaw beneath which the nerve was grasped; and he calculated that it was torn through only 1 or 2 cm. from the Gasserian ganglion. As to his results on the living subject, a marked degree of success attended his practice. Only one case had relapsed, and that in a very hysterical subject, although several of the operations had been performed six years previously. Paresis of the facial muscles had occurred temporarily in a few instances, and this is attributed to damage of the facial nerve terminals through the avulsion of the sensory twigs distributed with them.

The value of *nerve-stretching* as a means of treatment was accidentally discovered by Nussbaum, of Munich, in 1860, in consequence of the disappearance of a tetanic spasm of the arm after an accidental stretching of the ulnar nerve during resection of the elbow. In 1861 Hauser similarly by chance cured a contraction of the fourth and fifth fingers; and in 1869 Billroth,* unintentionally, cured a case of sciatica by the traction made during a thorough examination of the sciatic nerve. Nussbaum, however, reduced this plan of treatment to method, and first deliberately stretched the brachial plexus in 1874,† whilst Vogt, in 1876, appears to have been the first to use it in trigeminal neuralgia. Since that date the operation has been largely employed, and its value has been pretty accurately ascertained in the treatment of the many conditions to which it has been applied.

Its use in cases of neuralgia has not been very satisfactory, and especially is this so in facial tic. Statistics are not of much value in the investigation of this subject, inasmuch as the cases were not kept under observation long enough. Hahn, of Berlin, records eleven cases, of which eight relapsed after six or eight months, two were utter failures, and only one case was really improved, although not cured, and in this a portion of the nerve was excised after being stretched. Lagrange gives similar unsatisfactory accounts of the operation of stretching the supra-orbital nerve; out of fifteen cases there was but one known cure after three years, and five were quite unsuccessful; in one, there was but temporary relief, and the subsequent history of the other cases is not recorded. Of six neurectomies of the same nerve, four were successful.

* 'Archivs f. Klin. Chirurg.,' xiii, p. 379.

† 'Deutsche Zeits. f. Chirurg.,' 1874.

The same author contrasts neurectomy with nerve-stretching of the infra-orbital branch; whilst with the former 25·67 per cent. of the cases remained free from pain after three years, only 12·57 per cent. of those in which the nerve was stretched were free at at the same time.

Practically it is now admitted by almost all surgeons that nerve-stretching should be retained as a means of dealing with neuralgia of mixed nerves only, and undoubtedly it is beneficial in some such cases. But with the 5th nerve, which is almost entirely sensory, no permanent relief can be expected from the operation, and neurectomy is in every way to be preferred.

Probably, however, in all cases of neurectomy, a certain amount of stretching of both central and peripheral ends accompanies the manipulations necessary to free the trunks, and it is a perfectly open question as to whether or not such traction has any beneficial effect or otherwise on the course of the disease. That central changes can result therefrom is now a fact tolerably well established in those instances where the nerves pass directly through short, straight canals to the central organ. Thus Dr. Pauline Tarnowski, in the '*Archives de Neurologie*' (May and July, 1885), has reported the results of experiments made by him on rabbits. The sciatic nerves were stretched, and the spinal cords carefully examined at varying intervals of time. If slight traction alone was employed (*e.g.*, 500 grams), the only change found was a transient hyperæmia which soon passed away. But when the force reached 4 or 5 kilograms, more definite changes occurred; in the grey matter hæmorrhages and inflammatory exudations were seen soon after the injury, and later on sclerosis with atrophy of the nerve cells, such changes being always more marked in the posterior than in the anterior cornua. It is quite possible that similar effects may be produced in the Gasserian ganglion by stretching the branches of the second and third divisions, but evidently sufficient curative effect is not thereby established.

One other plan of treatment which has been suggested must be alluded to ere I bring this lecture to a close, viz., *ligature of the carotid artery*. It was originally recommended and practised by Trousseau; but up to the present time, so far as I can ascertain, only eighteen cases have been thus treated, and the results as given by Dr. Fowler have been as follows:—

In 4 cases, there was relief for over 3 years.

„ 3 „ „ „ „ 1—3 years.

„ 4 „ „ „ „ 1 year.

„ 1 „ partial relief.

„ 2 „ no relief.

One case was fatal. The longest period of relief was eleven years, but it must be noted that some of the reported cures had also been treated by nerve-stretching and excision. Roser is stated by Madelung* to have tied the external carotid for tic thrice, with one cure.

Of this plan I have had no experience, and, indeed, have heard of no cases in which it has been recently tried; but one cannot help being struck by the above results, especially in connection with the theory of obliterative endarteritis, alluded to earlier in this lecture, propounded by Professor Dana. The figures quoted indicate a measure of success which is certainly somewhat greater than that usually attending operations directed to the nerves themselves.

The mortality of the operation is certainly a serious objection to it, for, although it is less than when the artery is tied for aneurisms, &c., yet Hüter calculates it as about 5 per cent. in these cases, and such a fact would exclude it as a means of treatment, except as a last resort. Moreover, even if not fatal, the possible effect upon the cerebral hemispheres must not be lost sight of. That death has occurred from neurectomy is an undoubted fact, but this is usually explained by septic contamination of the wound, which can be avoided, and I am thankful to say that I have not yet lost a case in any of my nerve operations.† If, however, there should be a recurrence of the pain after dealing with the Gasserian ganglion this operation of carotid ligature can still be resorted to.

* ‘Archivs f. Klin. Chir.’ xvii.

† Since this lecture was written, I have to record one fatal case resulting from an operation on the Gasserian ganglion, for details of which see page 210.

LECTURE II.

1st Division.—Supra-orbital Nerve.—Supra-trochlear Nerve.

2nd Division.—Infra-orbital Nerve.—Removal of Meckel's Ganglion.

3rd Division.—Inferior Dental Nerve.—Gustatory Nerve.—Division of Trunk at Foramen Ovale.

MR. PRESIDENT AND GENTLEMEN,—It is my intention to devote this lecture to a consideration of the various operations on the different branches of the 5th nerve, passing each procedure rapidly under review. For the efficient performance of these it is essential for the surgeon to be well acquainted with the topographical anatomy of the face, and the various landmarks which serve as guides to the position of the nerves and arterial trunks. No apology, therefore, is necessary for introducing a brief anatomical sketch of each of the divisions of this nerve, previous to discussing the various operative methods.

The 1st or *Ophthalmic division*, after coursing along the outer wall of the cavernous sinus as a flattened band, enters the orbit through the sphenoidal fissure, dividing into three branches, the frontal, lachrymal, and nasal. The last two of these are of but slight surgical importance, and I shall not therefore allude to them. The frontal nerve, which forms the largest portion, divides into the supra-orbital and supra-trochlear, and both of these come within the domain of surgical treatment; consequently their exact position is of importance.

The *supra-orbital* nerve (Fig. 7, *a*) emerges from the orbit at the foramen or notch at the junction of the middle and inner thirds of the supra-orbital margin. It is accompanied by vessels derived from the ophthalmic artery and vein; the trunk of the nerve lies beneath the orbicularis palpebrarum, and is distributed to the forehead and anterior portion of the scalp. The notch, if present, forms a sure guide to the position of the nerve, which can be reached and exposed by a vertical incision immediately over it, as recommended by Sir William MacCormac, or by a transverse incision parallel to and a little below the eyebrow, as more commonly practised. The latter method leaves a less noticeable scar, whilst the former gives access to a larger portion of the nerve. Whatever the direction of the skin incision, the knife should afterwards be

carried in a direction parallel to the fibres of the orbicularis. The old subcutaneous operation is now seldom practised, being unsatisfactory, mainly owing to extensive extravasation from concurrent division of the supra-orbital vessels.

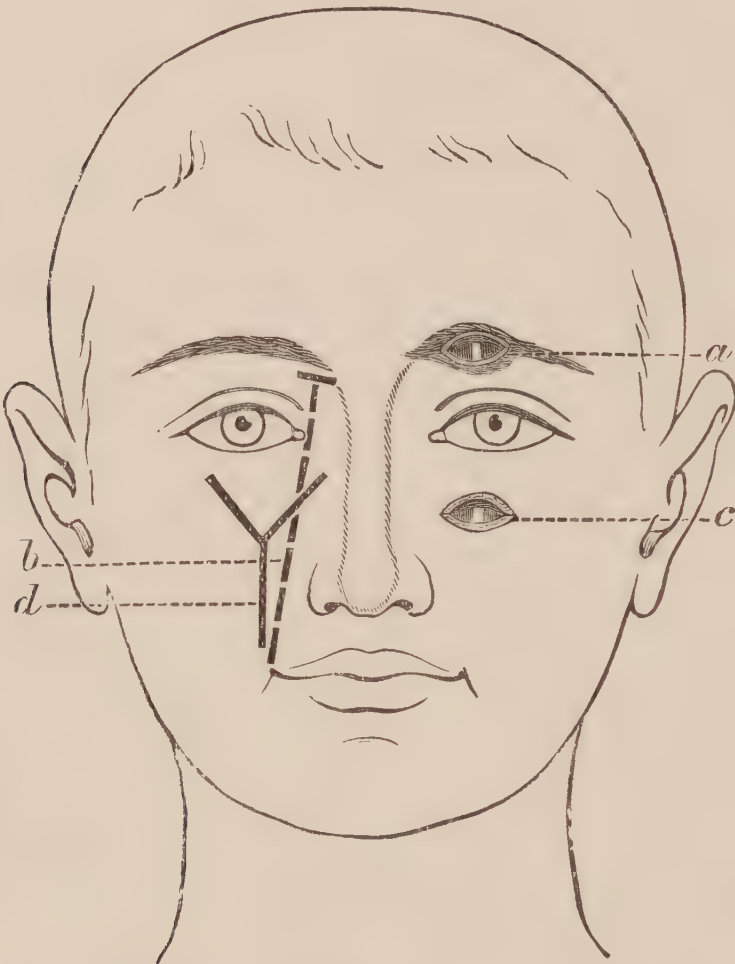


FIG. 7.—Diagram of full face, showing:—

- (a) Position of supra-orbital nerve, and incision exposing it;
- (b) Line indicating position of supra-trochlear nerve, passing from angle of mouth through the inner canthus. The short cross-line at its upper end is the position of the incision required to expose it;
- (c) Position of the infra-orbital nerve, and incision;
- (d) Carnochan's incision for neurectomy of the 2nd division.

When neurectomy is necessary, the orbit must be entered by dividing the orbito-tarsal ligament, the orbital fat depressed by a spatula, and the nerve freed from its connections and lifted on a blunt hook. It is divided as far back in the orbit as possible, and usually the supra-trochlear nerve is included in this procedure.

Occasionally the *supra-trochlear* nerve may demand treatment

separately. The guide to this is a line drawn upward and inward from the outer angle of the mouth through the inner canthus; the nerve is found where the prolongation from this line meets the orbital margin (Fig. 7, *b*). An incision parallel with the eyebrow over this spot and dividing the orbicularis will expose the fine filaments of the nerve, and they may then be dealt with as desired.

The *2nd* or *Superior Maxillary division*, after leaving the Gasserian ganglion and passing through the foramen rotundum, takes a directly horizontal course forward through the upper part of the speno-maxillary fossa, and onward under the floor of the orbit through the infra-orbital foramen. It is accompanied in its course by the infra-orbital artery, one of the terminal divisions of the internal maxillary; this vessel, often a trunk of some size, may give considerable trouble in operations in this region. The situation of Meckel's ganglion and the point where the posterior dental nerves come off is a little under 2 inches from the infra-orbital foramen; consequently to excise the former and divide the latter requires a somewhat deep dissection. The upper wall of the antrum is an important inferior relation, for the nerve is only separated by this from the antral cavity. The infra-orbital canal is in many skulls open above in its posterior half, constituting merely a groove; the anterior half inch is, however, invariably a true bony canal, which takes a somewhat downward as well as forward course. All the teeth of the upper jaw are supplied from this division; the molars, by the posterior dental branches arising close to Meckel's ganglion between it and the point of entrance into the infra-orbital canal; the bicuspid and canines are supplied by the middle dental offshoots which leave the main trunk in the hinder part of the canal, whilst the anterior dental branches supplying the incisors arise in the canal close to the infra-orbital foramen. An orbital branch is given off before Meckel's ganglion is reached, just external to the foramen rotundum, and this is distributed to the skin over the temporo-malar region, where neuralgia may be felt. The high origin of these nerves emphasises the importance of dealing with this division at any rate close to the point of emergence from the base of the skull.

Meckel's ganglion is a small mass of nervous tissue more or less triangular in shape, and about the fifth of an inch in diameter.

It lies in the spheno-maxillary fossa surrounded by the terminal divisions of the internal maxillary artery, and gives branches to the nose, pharynx, and palate.

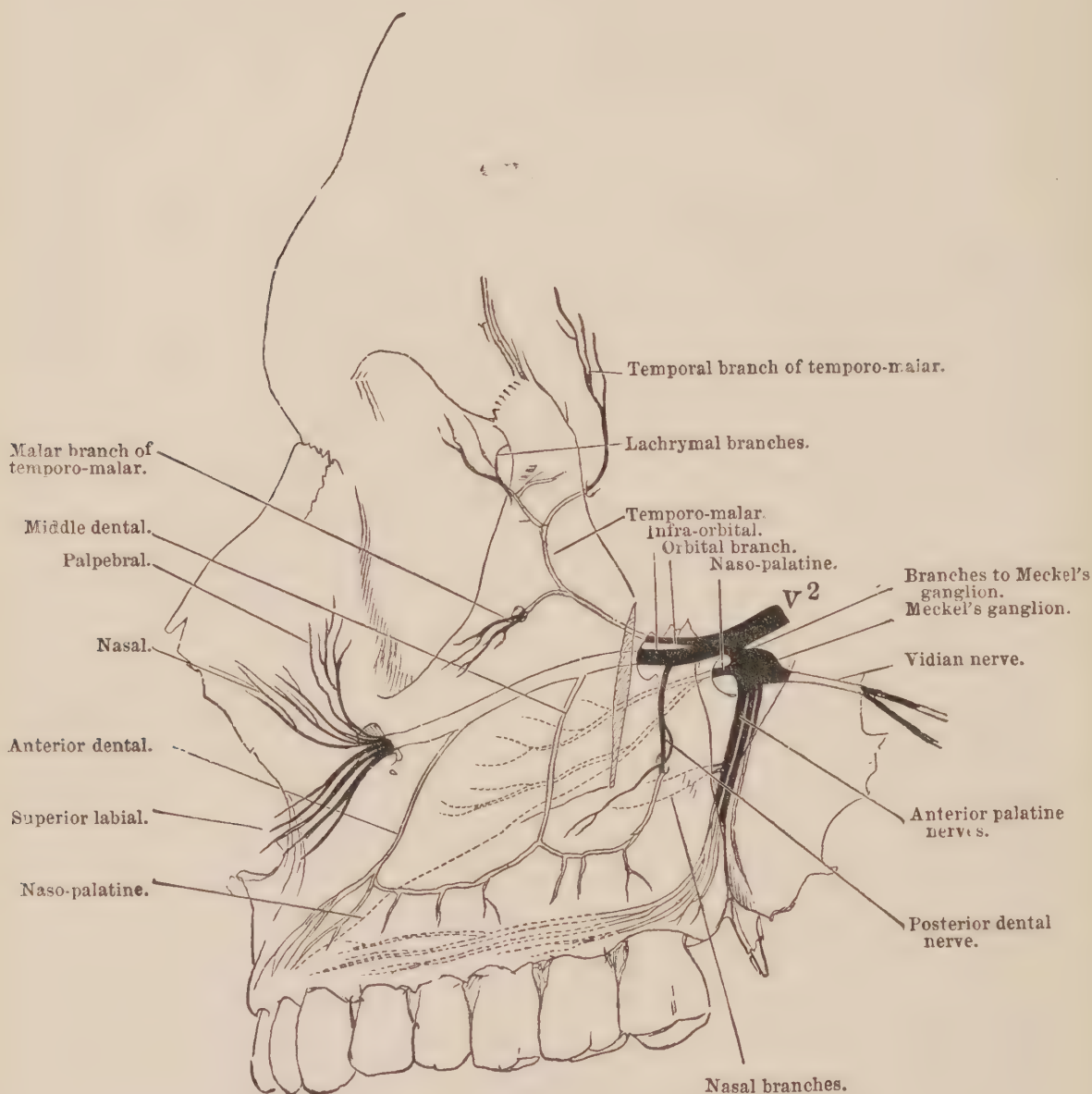


FIG. 8.—Distribution of the 2nd or superior maxillary division of the trifacial nerve. (*MacCormac.*)

The infra-orbital foramen is situated on a line drawn from the supra-orbital notch downward to a point between the two bicuspids. It corresponds to the junction of the inner and middle thirds of the infra-orbital margin, and is about half an inch below it. The nerve on emerging therefrom divides into palpebral, nasal, and labial branches, as shown in Fig. 6.

Formerly, the subcutaneous or submucous method of division at the infra-orbital foramen was most commonly employed; the surgeon simply passed a tenotome into the foramen, and twisted it round. I well recollect on one occasion more than twenty years ago, a celebrated surgeon accidentally breaking his knife off short and leaving the point in the foramen, with the remark that the steel might act as a nerve tonic, a result which, unfortunately, was not realised.

To expose the foramen, all that is needed is a transverse incision extending through the orbicularis and separating its fibres. The margins of the wound must then be held apart with blunt hooks or spatulæ, and by a careful dissection the nerve is seen emerging from the foramen. Half an inch of it can be removed by this procedure. (Fig. 7.)

In order to expose and excise larger portions of this nerve, three distinct plans have been suggested and practised, which may be named respectively, the *orbital*, the *antral*, and the *pterygo-maxillary*.

(A.) The *orbital* method.

Several different operations may be included under this heading, notably Langenbeck's and Wagner's. In the former, the infra-orbital foramen is first exposed by a transverse incision, and the nerve freed at its point of exit, and secured by forceps. Dieffenbach's tenotome, a narrow-bladed, slightly curved or hooked knife, is now entered, with its cutting edge directed downward close to the external canthus, just below the outer palpebral ligament, and is passed backward toward the apex of the orbit along the anterior border of the pterygo-maxillary fissure, which is crossed by the nerve about an inch behind the orbital margin. Care must be taken to keep it close to the bone, and not to insert it too far, lest the spheno-maxillary fossa be entered and serious hæmorrhage result. By now withdrawing the knife carefully, and keeping its edge closely applied to the bone the nerve can be divided as it enters its canal. Traction or torsion upon the peripheral end will enable the whole length of the nerve to be removed and the dental branches given off it in its course to be torn through. Malgaigne* made a similar preliminary incision, but divided the nerve as it entered the canal by passing a tenotome

* 'Manuel de Méd. Opérations,' 4th edition, p. 153.

along the floor of the orbit. The great objection to both of these proceedings is the hæmorrhage which results from an indefinite division of vessels in a situation inaccessible for ligature, and the uncertainty which necessarily attends the use of a knife at such a depth when the surgeon is, so to speak, working in the dark. In the present day with the confidence gained by experience in the use of antiseptics, open operations should usually be adopted in preference to subcutaneous methods, which latter must of necessity be uncertain, and lacking in precision, and especially so when the structures to be divided are in dangerous localities.

With a view of obviating this, Wagner introduced a different plan. He raised the orbital contents from an incision running transversely below the orbital margin, laid open the infra-orbital canal with a chisel, separated the artery from the nerve with a special aneurism needle having a lateral curve, and divided the nerve *behind* the origin of the dental branches. It appears to me that this operation could be conducted with greater facility if more space were obtained by a freer use of the chisel, trephine, and other suitable instruments; and considering the close proximity of Meckel's ganglion, it is better surgery to extend the operation to its removal, as the gain will probably be more decided without any considerable increase in its severity. The value of this additional proceeding is evidenced statistically by the report given by Dr. Fowler, of Brooklyn,* who records 83 cases dealt with, and contrasts the results of 26 in which the ganglion was removed with those of 26 in which it was left behind. In the former series where the ganglion was removed, three patients obtained relief for more than 3 years, six for 2-3 years, nine for 1-2 years, and eight for under 12 months. Of the 26 where the ganglion was left, five obtained relief for over 3 years, three for 2-3 years, seven for 1-2 years, and eleven for under 12 months, and he calculates that the average period of relief was 1 year, 5 months, and 16 days when the ganglion was removed, and 1 year, 3 months, and 15 days when the ganglion was left intact. From these statistics the gain derived from removal of the ganglion is not so great as we should expect, but yet one cannot but feel that it is correct practice to deal with it by extirpation. I feel it must be admitted that all operations for the relief of tic of the 2nd division of the 5th nerve are unsatisfactory.

* 'Annals of Surgery' (St. Louis), iii, 1886, p. 269.

In his recent paper Professor Horsley* has described his plan of operating which is practically the same as Wagner's. The eyelids are stitched together as a preliminary precaution. His incision is a semilunar one along the inferior orbital margin, combined with a vertical one placed at right angles to it over the infra-orbital foramen, and about three-quarters of an inch in length. The flaps thus marked out are raised from the bone, the periosteum being included. The orbital tissues are now freed from the bone, and emphasis is laid on the fact that if the orbital periosteum be maintained whole and unbroken, the orbital fat is not seen and does not protrude into the wound. The infra-orbital canal is laid open with a fine pair of bone forceps, and as a rule the antrum remains intact. Should it be accidentally opened its cavity is to be filled with powdered boracic acid, and no interference with the healing of the wound is to be expected. The nerve is freed as far as the foramen rotundum and there divided.

(B.) The *antral* method was devised and originally introduced by Carnochan,† of New York, in 1858, in order to effect removal of Meckel's ganglion, which he considered to be of great importance. Twenty-two cases of the operation were collected and brought before the Royal Medico-Chirurgical Society of London in 1884 by Chavasse, who had himself performed the operation several times, introducing slight modifications in the technique.

Carnochan's incision was V-shaped with the apex over the infra-orbital foramen pointing downwards (Fig. 7, *d*); each limb of the V should be about 1 inch long. The flap thus marked out is turned up and held out of the way, and a vertical incision made from its apex to the angle of the mouth opening into the oral cavity. This permitted the infra-orbital nerve to be well defined and set free, and the anterior wall of the antrum clearly seen.

Chavasse proposed the use of a T-shaped incision, the transverse limb being made parallel to the fibres of the orbicularis, and the vertical limb extending nearly to the angle of the mouth, but not opening it. Sufficient room is gained by this means; but for the later steps of the operation an efficient electric incandescent lamp is most essential, and a small hand-lamp in a bell-shaped reflector held by an assistant in any position the operator may desire will best serve this purpose; some surgeons, however,

* Horsley, 'Brit. Med. Journ.,' Nov. 28th, 1891.

† 'Amer. Jour. of Med. Sci.,' 1858, p. 136.

prefer the lamp to be fixed to the forehead. The anterior wall of the antrum is next opened, either by a $\frac{1}{2}$ -inch trephine, or with a chisel and mallet, as suggested by Mr. Treves, the mucous lining torn through, and the cavity fully exposed. The posterior wall of the antrum is perforated in a similar way, either by a $\frac{1}{4}$ -inch trephine (Chavasse) or by a chisel (Carnochan). The sphenomaxillary fossa is thus reached and profuse hæmorrhage must be expected, which should be checked as far as possible by sponge pressure. The infra-orbital canal is then opened along the roof of the antrum by incising the mucous membrane, and picking away the bony walls of the canal with a chisel or fine bone forceps. If the nerve be seized in a pair of catch forceps, this proceeding will be facilitated; and thus the nerve can be gradually traced back into the fossa, and up to the foramen rotundum. At this stage the infra-orbital vessels usually give trouble. The trunk of the nerve can now be divided close to the foramen by a pair of fine curved blunt-pointed scissors; Meckel's ganglion is defined lying a little below the nerve in the fossa, and removed; and the posterior dental branches are torn or cut through. It is not always easy to see the ganglion, and occasionally its removal is from this cause a matter of guess-work, for the hæmorrhage may be so severe as to prevent any clear vision of the parts. Bleeding having been arrested by sponge pressure, &c., the wound is thoroughly washed out and closed; a small drainage tube can be inserted with advantage into the lower angle of the V.

The results of this operation are not, on the whole, encouraging, for in Chavasse's collection of twenty-two cases, only three seem to have derived permanent benefit. Of five cases similarly treated by Mr. Treves, one experienced a recurrence of the neuralgia at the end of three years, one at the end of two years, two within twelve months, whilst the fifth died of cancer within six months.

Von Bruns* and Otto Weber† modified this operation of Carnochan's by making a semilunar incision along the infra-orbital margin, raising the orbital tissues, freeing the infra-orbital nerve at its foramen, and then with a fine saw cutting in a circular direction around the foramen at a distance of half a centimetre. This must extend backwards as far as the sphenomaxillary fissure,

* Von Bruns's Atlas, Abth. II, Taf. xv, Fig. 10.

† Otto Weber, *op. cit.*, p. 166.

and the bone must be completely detached so that it may be slipped over the nerve, and thus the fossa freely opened. The remaining steps of the operation are the same as in Carnochan's.

(C.) The third means of dealing with this division of the 5th nerve is the *pterygo-maxillary operation*, originally practised by Professor Lücke.* He made an oval incision extending from the outer canthus first backward, and then downward and forward; the masseter muscle was then detached from the lower border of the zygoma, and turned down, whilst the zygoma itself was sawn through in front and fractured behind, so that it, together with the temporal fascia attached, could be turned upwards. By carefully dissecting down in front of the tendon of the temporal muscle the nerve was reached and divided as it crossed the spheno-maxillary fossa, the parts were then replaced, the masseter being sutured to the zygoma. The results of this method of dealing with the zygoma were extremely unsatisfactory, from the fact that the muscle usually did not unite with the bone, and hence cicatricial deformity and functional weakness were produced. Professors Braun† and Lossen‡ have introduced a plan to obviate this by detaching the temporal fascia from the bone, and turning the latter downward together with the masseter. The incision employed is angular (Fig. 9, *a*), starting from just behind and below the external angular process of the frontal bone backward to the tragus, and downward and forward into the cheek. A flap of skin and subcutaneous tissue is reflected; the zygoma divided in front and behind, and turned down; the tendon of the temporal muscle is drawn backward with the mouth slightly open. The pterygo-maxillary fissure is in this way exposed, and the nerve sought for in the fossa and divided (Fig. 10). This procedure removed one of the objections to reaching Meckel's ganglion by this route, but in order to remove the infra-orbital nerve it is necessary to make an additional incision over the site of the infra-orbital foramen (Fig. 10, *d*). The great advantages which are claimed for this operation are that the cicatrix will be less obvious on looking at the patient full-face, and the hæmorrhage will be diminished in that the internal maxillary artery can be secured before the fossa is reached. Moreover, the antrum is not opened,

* 'Deut. Zeitschrift f. Chirurg.,' June 9, 1874.

† 'Cent. f. Chirurg.,' 1882, No. 16.

‡ 'Cent. f. Chirurg.,' 1878, pp. 65 and 148.

and hence the risk of septic contamination of the wound is considerably lessened.

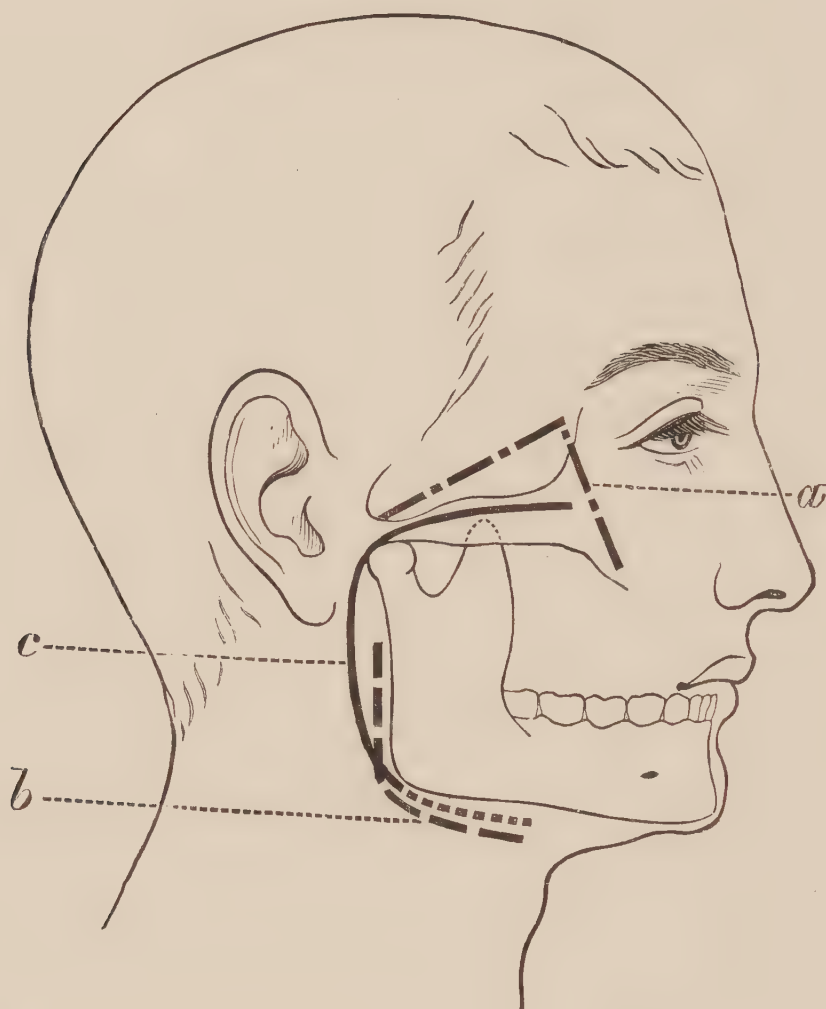


FIG. 9.—Diagram of side face showing:—

(a) Incision in the Braun-Lossen method of reaching the 2nd division of the 5th nerve at the foramen rotundum;

(b) Lücke-Sonnenburg incision for exposing the foramen ovale;

(c) My own incision for deepening the sigmoid notch. The dotted extension represents the additional incision needed in operating for the Gasserian ganglion.

Since writing the above I have had occasion to operate upon a case of long-standing ankylosis of the lower jaw, for which I thought it desirable to excise the coronoid process on the affected side. The preliminary steps of the operation were very similar to those detailed above, with the addition that I drilled the zygoma before and behind for subsequent wiring, and made use of a

different skin incision. After removal of the coronoid process as well as division of the deep fibres of the temporal muscle and forcible depression of the lower jaw, I took the opportunity of

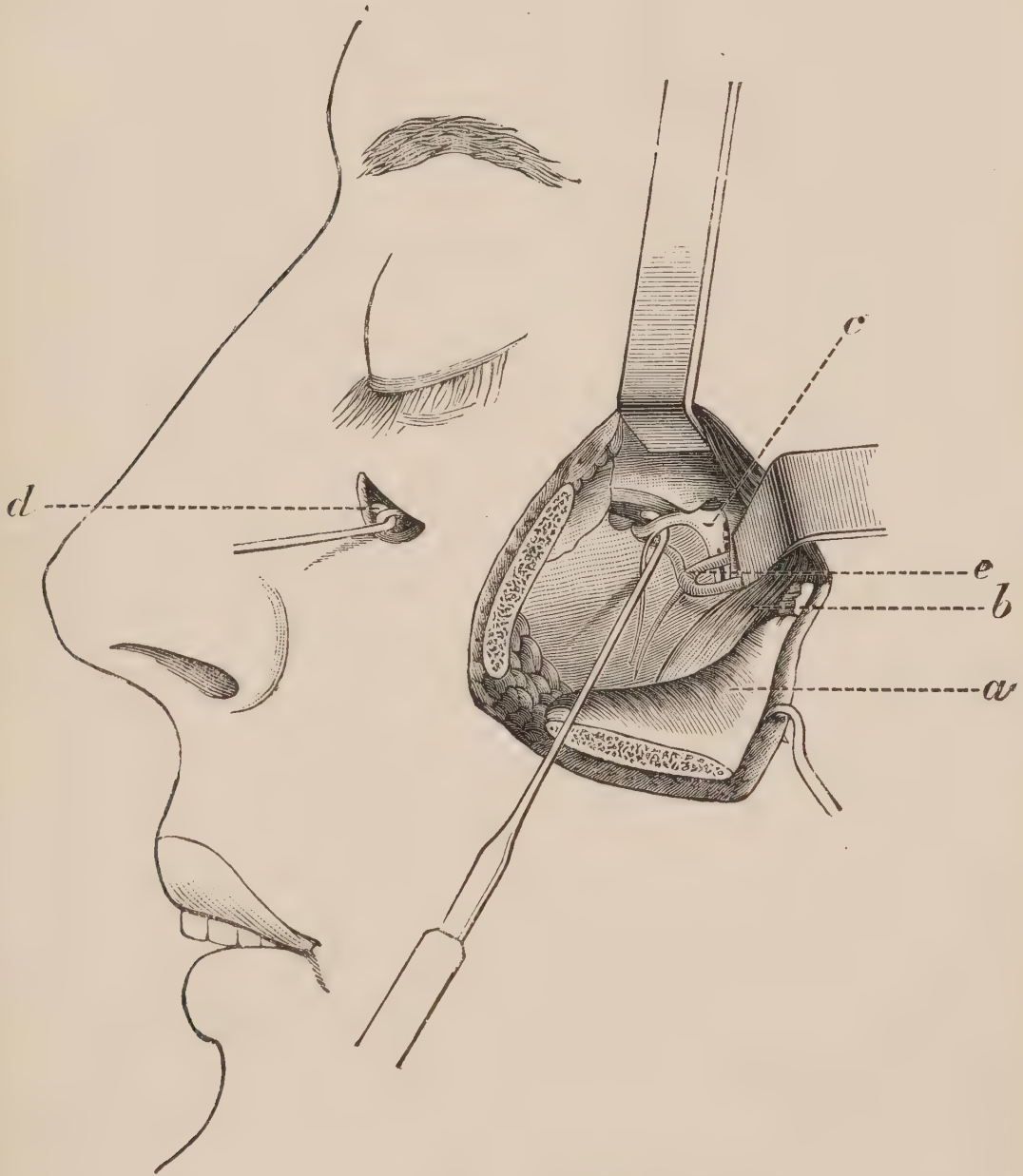


FIG. 10.—Diagram showing dissection necessary to expose the 2nd division of 5th nerve, according to the Braun-Lossen method.

- (a) Zygomatic arch divided and turned down;
- (b) Temporal tendon, arising from coronoid process, and held back by retractors;
- (c) Superior maxillary nerve and Meckel's ganglion;
- (d) Infra-orbital nerve at the emergence from the canal.

examining the practicability of this operation, and found that the trunk of the nerve could be readily hooked up on an aneurism needle passed through the pterygo-maxillary fissure into the spheno-maxillary fossa. I have also found it easy to demonstrate this on the cadaver. I certainly think that this is a most direct and valuable method of reaching the nerve at the foramen rotundum if it be desirable to divide it at that spot. Eleven cases of this operation have been collected by Segond,* and the results reported in 1890. Czerny† had performed five cases; one recurred, but with less intensity, in nine months; one had a temporary relapse after ten months, and the other three were reported well at four months, nine months, and over two years respectively. Grisson‡ records three cases in Madelung's clinic reported well at five months, two years, and two years and a half; and Segond himself reports one case well after one year, and two other cases too recent to be of statistical value.

THE 3RD DIVISION.

The *3rd or Inferior Maxillary division* of the 5th nerve as it emerges from the base of the skull through the foramen ovale is joined by the motor root, and divides immediately into two trunks. The anterior or smaller division consists almost entirely of motor fibres, and splits up into muscular branches to the pterygoids, temporal, and masseter, the small buccal branch being probably sensory in function. The posterior division, which is almost entirely sensory, divides into three branches, the auriculo-temporal, the lingual, and the inferior dental (Fig. 11). The two latter are chiefly of surgical importance, and we must now proceed to discuss both their anatomical relations and the surgical procedures directed towards their trunks, reserving to the end of this lecture the means of dealing with the 3rd division in its entirety at the foramen ovale.

THE INFERIOR DENTAL NERVE.

The *Inferior Dental Nerve* is the largest of the three branches of this division. It passes down from the foramen ovale beneath the

* 'Revue de Chirurg.,' 1890, March 10th.

† 'Deutsche Zeitschrift f. Chirurg.,' 1882, No. 16.

‡ 'Berl. Klin. Wchshrtt.,' 1887, Dec. 21.

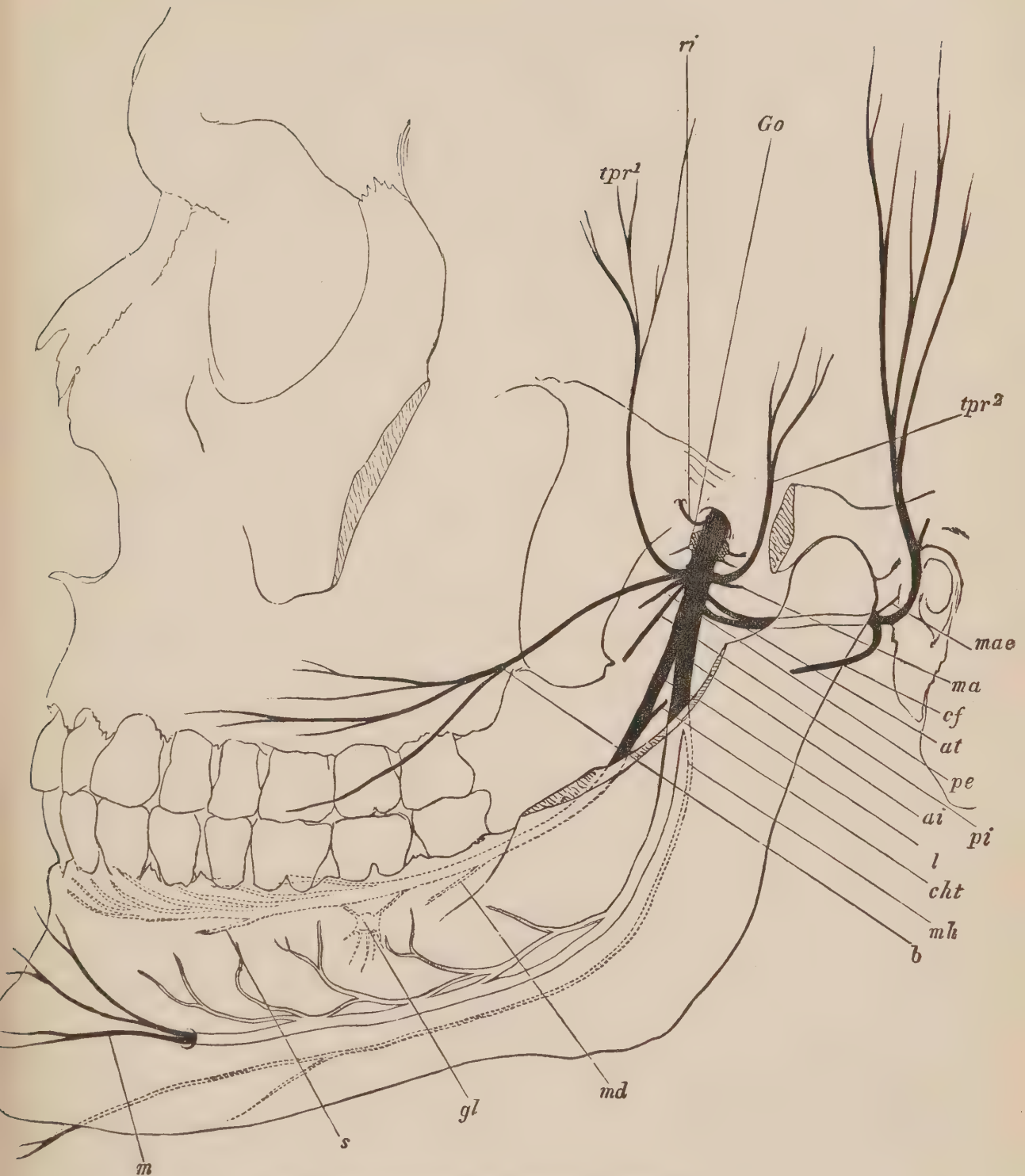


FIG. 11.—3rd division of the 5th nerve. (MacCormac.)

*tpr*¹. Anterior temporal branch.

ri. Recurrent branch.

Go. Otic ganglion.

*tpr*². Posterior temporal branch.

mae. Branch to meatus auditorius externus.

ma. Masseteric branch.

cf. Communicating branch to facial nerve.

at. Auriculo-temporal.

pe. Nerve to external pterygoid.

pi. Nerve to internal pterygoid.

ai. Inferior dental.

l. Lingual.

cht. Chorda tympani.

mh. Mylo-hyoid branch.

b. Buccal branch.

md. Branches to the mucous membrane.

gl. Submaxillary ganglion.

s. Branches to the submaxillary gland.

m. Mental branch.

external pterygoid muscle, and then lies between the internal pterygoid and the vertical ramus of the jaw. When it reaches the dental foramen it is joined by the dental artery, a branch of the internal maxillary, and together they pass into and through the canal, the artery being superficial or external to the nerve. After supplying the molar teeth, it divides into two terminal branches, the incisor and the mental, the latter emerging from the mental foramen to be distributed to the skin of the chin, and communicating with the facial nerve in the substance of the orbicularis oris. Previous to entering the dental canal, a small muscular branch is given off to supply the mylo-hyoid and the anterior belly of the digastric muscles.

It may be exposed in three situations, viz., at its exit from the mental foramen, in the dental canal, and above the dental canal.

At its exit from *the mental foramen* it may be divided by a sub-mucous operation. Its position is easily ascertained, viz., below and between the bicuspid teeth. The operation, however, is not to be recommended, as the pain invariably returns.

The nerve can be reached *in the dental canal* by removing a disc of bone comprising the outer layer of the jaw by means of a three-quarter inch trephine applied over its course about the junction of the ascending and horizontal rami. If the disc be skilfully removed, the nerve and the artery can be seen running together in the bone, and the former lifted up on an aneurism needle, whilst it is well to divide the latter between a double ligature. The skin incision should be made along the line of the jaw, and about $\frac{1}{2}$ inch behind it, never over the part to be trephined. By this means the cicatrix is less evident, and the wound in the skin does not correspond with that in the bone.

Another somewhat similar method of dealing with the nerve is that of Velpeau, who through the same kind of incision trephined the jaw through the ascending ramus, immediately over the dental foramen. At this spot the bone is much thicker below than above, and hence the trephine should not be used when the upper part of the jaw has been divided; the remaining half circle of bone is best detached by means of an elevator and chisel.

Linhart operates in much the same way, but detaches the masseter by a vertical incision which is liable, I should fear, to divide all the branches of the facial nerve in that locality, and consequently lead to paralysis and subsequent facial disfigurement.

Gross has recommended the use of the dentist's drill as a means of destroying the nerve in the canal by perforating the bone over the course of the dental canal, thus cutting the nerve across.

These operations are extremely easy, and the wounds heal without trouble. The results, however, are not lasting, the pain returning after an interval varying from a few months to a year. I have, therefore, entirely abandoned this method of treating cases of neuralgia of this nerve, and always have recourse to higher operations.

The inferior dental nerve has been attacked *just above* its entrance to *the dental canal* in the lower jaw by three different methods, which we may fairly describe as—

- (a) the intra-buccal method ;
- (b) the retro-maxillary ; and
- (c) the trans-maxillary.

The *intra-buccal* method is the form the operation originally took ; it was proposed by Lizars, and performed for the first time by Michel (of Nancy) in 1856. More recently it has been again advocated by Billroth, but it is commonly known as Paravicini's operation.

The nerve lies close under the mucous membrane of the mouth for about an inch of its course after getting clear of the internal pterygoid muscle, and then runs between the internal lateral ligament of the temporo-maxillary joint on the inside, and the ramus of the jaw externally. The gustatory nerve pursues a parallel course a little internal and anterior to it. The dental foramen is guarded at its anterior and inner aspect by a sharp osseous projection, the *lingula* or spine of Spix.

The intra-buccal operation is performed as follows :—

The mouth is opened with a gag, preferably unilateral, placed between the molar teeth on the opposite side. The cheek on the affected side is held well out of the way with retractors, and, if necessary, the tongue pulled to the opposite side with forceps. It is now tolerably easy to define the entrance to the canal, and feel both nerve and artery rolling under the finger. The spine of Spix cannot be so clearly distinguished as on the skeleton from the fact that it is obscured by the attachment of the internal lateral ligament. The mucous membrane is incised over the anterior border of the ascending ramus of the jaw in a vertical direction

for about 1 inch, and this incision must be carried down to the bone. The soft parts are next detached from the jaw, and in order to reach the orifice of the canal the internal lateral ligament will need division; this may be done by means of a fine pair of blunt-pointed scissors, such as are used in strabismus operations. The nerve can now be seen, and isolated from the vessels; it is freed from its connections as far up as possible, and divided, care being taken not to wound the internal maxillary artery by going too high. It is again divided close to the foramen, and the intervening portion removed; this will only consist of about half an inch. Of course a good electric light is absolutely essential.

This operation is admittedly objectionable from many points of view. In the first place, we have a wound communicating with the mouth, which is always undesirable, and one of Michel's cases died of septicæmia, as a result. Then, again, only a very limited portion of the nerve can be excised (Billroth says 1 inch), and, moreover, from the cramped space the proceeding is very difficult. Hæmorrhage from a wound of the inferior dental artery is not an uncommon accompaniment, and when it occurs it is not easy to deal with, from the inaccessibility of the parts; in consequence of this, the external carotid has, I understand, required ligature more than once.

The *retro-maxillary* operation, or as it is commonly called, the Lücke-Sonnenburg method, after the two surgeons who originated it, is described by Ullman, who has recently advocated its use as follows:—

The head is allowed to rest on the sound side or even hanging over the end of the operating table. An incision is made along the posterior border of the jaw, and from 1·5 cm. above the angle to the centre of the horizontal ramus (Fig. 9, c). The branches of the facial nerve to the neck are the only ones of importance divided, but the facial vessels will of necessity be divided and need to be ligatured. The parotid fascia is next incised, and the gland drawn upward as far as possible. The internal pterygoid muscle is detached by scissors from the mandible, and the entrance to the inferior dental canal is indicated by the spine of Spix which should be carefully sought for. The nerve is isolated and divided with scissors peripherally; it is held tense with forceps, and serves, if necessary, as a ready guide to the foramen ovale.

The *trans-maxillary* operation is that which promises to be the

most effectual, and is, perhaps, that most generally adopted in the present day. It is usually accomplished by deepening the sigmoid notch. Inasmuch, however, as the lingual nerve can also be dealt with by this method, and since both nerves usually need simultaneous division close to the base of the skull, it will be well to defer the description of this operation till we deal with the different methods of reaching the trunk of the nerve at the foramen ovale.

THE LINGUAL NERVE.

The *lingual nerve* lies in the first place beneath the external pterygoid muscle together with the inferior dental, but is internal and a little anterior to it. It then passes between the internal pterygoid muscle and the inner side of the vertical ramus of the lower jaw, crossing obliquely above the superior constrictor of the pharynx to reach the side of the tongue. In this part of its course it lies immediately beneath the mucous membrane of the mouth, and can be readily felt at a point corresponding to the junction of the middle and upper thirds of a line drawn between the angle of the jaw and the last molar tooth. It thence courses forward to the apex of the tongue lying superficial to the hyoglossus muscle, crossing Wharton's duct.

It can be easily reached through the mouth by incising the mucous membrane if it be thought desirable. The cheek must be held aside by a broad retractor, and the mouth kept open by an efficient gag. Sir William MacCormac recommends the division of the cheek also, but this is surely an undesirable addition, particularly as the operation is itself usually ineffectual. The nerve is seen passing downward and forward, and can be raised on a blunt hook, and stretched or divided. About one inch of it may be removed in this manner. There are many objections to this proceeding; in the first place, the nerve is reached at some distance from its exit from the skull; secondly, troublesome hæmorrhage may obscure the part, and render the operation very difficult; and lastly, it is impossible to keep the wound aseptic. Although I used to practise this method in my earlier cases, I have long discontinued it in favour of the external operation by which means perfect asepsis can be maintained, and the trunk of the nerve divided close to the base of the skull. Moreover, the branches of the inferior dental are frequently affected as well, so

that it is better to deal with the two together by the operation of deepening the sigmoid notch. In cases of malignant disease of the tongue where the pain is very severe, it may be occasionally useful to reach and divide the nerve in this way through the mouth; but as a rule more extensive operative procedures are necessary.

It has also been suggested by Luschka to reach the lingual nerve through the submaxillary region by an operation very similar to that for tying the lingual artery. An incision is made along the lower border of the horizontal ramus, the cervical fascia divided, and the submaxillary gland drawn forward and downward. The digastric will thus be exposed lying anteriorly upon the mylo-hyoid, and held down by fascial attachments to the body of the hyoid bone. The hyo-glossus muscle is also seen, extending vertically upwards, and the lingual nerve crossing its outer surface at its highest part can there be divided. The depth at which the nerve lies is the greatest difficulty in dealing with it in this way, and is a serious bar to the adoption of the operation. Even when the nerve is reached, only a small portion can be removed, and that at a considerable distance from the foramen ovale.

The nerve can also be reached by the Lücke-Sonnenburg method described above, the inferior dental being included in the same operation, if necessary; and Löbker has operated by making a curved incision along the antero-inferior border of the masseter muscle, and then by chiselling away the anterior border of the ascending ramus of the lower jaw, the lingual nerve can be seen and dealt with, lying on the outer surface of the internal pterygoid.

The want of success attending these methods of dealing with the peripheral branches has led surgeons to extend the scope of their operations to the base of the skull, hoping by a division of the nerve at its point of emergence, and by a more extensive removal of the nerve trunks, to obtain results of a more permanent character.

DIVISION OF THE NERVE AT THE FORAMEN OVALE.

Previous to describing the different methods of reaching the foramen ovale, which have been proposed and practised, it may be well to examine its chief anatomical relations.

Surgical Anatomy.—The foramen ovale is an aperture at the

root of the great wing of the sphenoid, close to its junction with the external pterygoid plate. It lies immediately behind and external to this process, and is on a level with the eminentia articularis of the temporo maxillary joint, being placed at a depth of about $1\frac{1}{2}$ –2 inches from the surface of the face (Fig. 12, F.O.). The long axis of the foramen is directed backward and out-

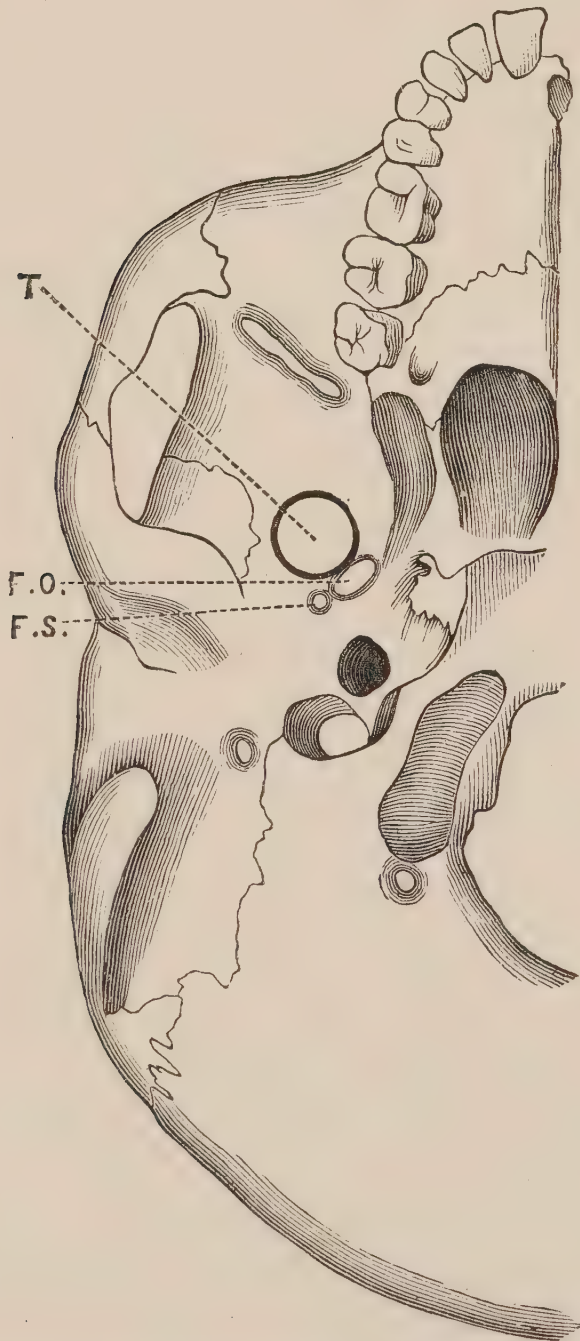


FIG. 12.—Diagram of base of skull, showing relations of the foramen ovale.
F.S. Foramen spinosum. F.O. Foramen ovale.

ward, and its outer margin is only separated by a narrow neck of bone from the foramen spinosum which lies at the base of the sphenoidal spine, and through which the middle meningeal artery passes. It will be seen, therefore, that the foramen ovale lies midway between the base of the external pterygoid plate and the spine of the sphenoid, being covered by the origin of the external pterygoid muscle. The internal maxillary artery is superficial to both pterygoid muscles in the majority of instances, lying between the external pterygoid and the jaw, and passing into the sphenomaxillary fossa through its two heads. Occasionally the vessel passes between the two muscles, and thence to the fossa. This point should be kept in view in any operation upon these parts, as the early ligature of the vessel is most desirable. There is also a large plexus of veins in this region, from which troublesome hæmorrhage is sure to arise. But, however variable the vascular distribution, the dental and gustatory nerves are quite regular in the position which has been already described; emerging from beneath the external pterygoid muscle, they lie upon and are superficial to the internal. To expose these nerves in some way or other must be the first aim of the surgeon, and this done they can be traced to the base of the skull with comparative ease, and there divided.

Three different plans have been suggested and practised for reaching the foramen ovale, viz.:—(α) Pancoast's method, with various modifications as suggested by Salzer, Krönlein, &c.; (β) the Lücke-Sonnenburg method; and (γ) the plan of deepening the sigmoid notch as practised by Mr. Horsley, myself, and others.

(α) *Pancoast* was the pioneer in this department of surgery, and his first operation was performed nearly twenty years ago. It appears that only one case was treated in this way, but the operation was an important step, and is justly deserving of record, in that it has served as a basis upon which many modifications have since been grafted. He dissected up a rectangular flap composed of skin and masseter muscle with its base at the zygoma, and exposed by this means the temporal tendon which was then detached from the coronoid process. This process was next removed by cutting pliers close to the ramus of the jaw, and the pterygo-maxillary fossa opened up. The attachment of the external pterygoid muscle was then divided, the internal maxillary

artery having been, if possible, previously tied. The third division of the trigeminal could thus be reached as it emerged from the foramen ovale. The operator must, however, have been considerably cramped in his manipulations, as the zygoma was left intact. Its removal or temporary displacement was soon added as an additional step in the operation. Again, the incision was devised irrespective of the distribution of the facial nerve, and the integrity of the duct of Stenson must have been seriously threatened. Of necessity the resulting cicatrix was extremely unsightly.

Salzer operated through a curved incision, having its convexity upwards; it extended from just in front of the tragus to a point above the zygoma, and terminated anteriorly at the lower border of the malar bone. This incision passed directly to the bone, and the zygoma was divided by the saw anteriorly at its attachment to the malar, and posteriorly in a line with the eminentia articularis. The temporal muscle was divided by a transverse incision across its fibres a little above the zygomatic arch, and the whole flap of soft tissues, including the loose piece of bone, was drawn down by scraping away with a raspatory the lower fibres of attachment of the temporal muscle to the squamous bone, until the pterygoid ridge of the great wing of the sphenoid was reached. Still keeping close to the bone, by detaching the external pterygoid muscle access was gained to the foramen ovale; this was much facilitated by opening the mouth, as by this means the coronoid process was depressed, and kept out of the way. In this procedure the operator has to work down on the nerve from above, and his manipulations are much hampered by the limited space at his disposal. Necrosis of the zygoma has, moreover, resulted, but by taking suitable precautions this can be prevented. If it does occur it will always leave an unsightly cicatrix.

Another modification of Pancoast's original plan is that devised by Krönlein* in order to reach and divide both the second and third divisions of the nerve at the base of the skull. This method, with the exception of the skin incision, is practically identical with the preliminary steps of my present operation for removing the Gasserian ganglion, which will be described in my next lecture. In brief, it consists in dividing the zygoma in front and behind, and turning it down with the masseter after releasing it from the

* 'Deutsche Zeitschrift für Chirurg.,' xx, p. 484.

temporal fascia; and in dividing the coronoid process with the chisel and turning it up together with the temporal tendon. The external pterygoid muscle is then detached from the skull and the foramen ovale exposed. The foramen rotundum is reached afterwards through the pterygo-maxillary fissure, somewhat according to Lücke's method already described. The same objection applies to this as to the former operation in that it is an unnecessarily severe proceeding when the third division alone is to be dealt with; a similar result can be obtained by the simpler operation of deepening the sigmoid notch. But if it be requisite to divide the second division simultaneously with the third, then this plan may be advantageously employed. Such a step, however, will be rendered unnecessary if the Gasserian ganglion can be safely reached and dealt with.

(β) The foramen ovale has also been reached by the Lücke-Sonnenburg or retro-maxillary operation. Ullman* has recently recorded two cases treated in this way, and speaks most highly of it, inasmuch as the bones of the face are not interfered with except that it is occasionally necessary to divide the angle of the jaw and turn it temporarily outwards, replacing and wiring it subsequently (as suggested by Albert); the superficial nerves divided are of no importance, the scar is placed well out of view, and the subsequent movements of the jaw are less likely to be impaired.

Mikulicz and Obalinski have proposed to reach the foramen ovale in a somewhat similar way. The former makes an incision from the mastoid process along the anterior border of the sterno-mastoid to the level of the hyoid bone, and then upward and forward to the border of the jaw. The skin is now dissected up, and the bone, carefully stripped of its periosteum, is divided by a chain saw behind the wisdom tooth, great care being taken not to open the mouth cavity. The internal pterygoid muscle is next to be detached, and the vertical ramus can then be drawn well outwards leaving a funnel-shaped opening with its apex at the base of the skull in which the nerves are readily found and traced to the foramen.

(γ) The third method and that which I consider the best, not only for reaching the foramen ovale, but also for removing the largest extent of these trunks, is that of *deepening the sigmoid notch*, a proceeding modified by Mr. Victor Horsley from Velpeau's

* 'Wiener Klin. Wochenschrift,' June 20th, 1889.

original method of trephining the jaw. The following description of the way in which I now perform this operation is similar to Mr. Horsley's, with a few exceptions, and, indeed, this method was originally suggested to me by him.

The skin is first rendered perfectly pure by previous washing with 1-20 carbolic lotion, and any hair or down removed, the razor being carried for a short distance into the temporal region. The auditory meatus and external ear are purified thoroughly, and the former plugged with a piece of salicylic wool or cyanide gauze. The skin incision is so planned as to leave a scar as unobtrusive as possible. Commencing about the middle of the zygoma, the knife is carried backward and downward over the parotid region to the angle of the jaw, and then for a short distance along the horizontal ramus (Fig. 9, c). A semilunar flap consisting of skin and

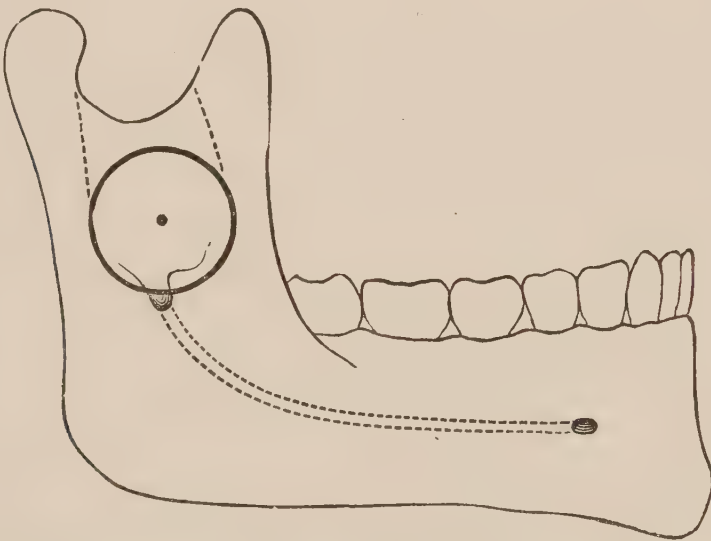


FIG. 13.—Side view of lower jaw, showing position of trephine opening in the operation for deepening the sigmoid notch. The two upper dotted lines indicate the extent of the bridge of bone, which also needs removal.

subcutaneous tissue only should be raised and turned forward, and for convenience temporarily stitched across the opposite side and carefully protected. This flap must be so dissected as not to injure any of the branches of the facial nerve. By this means are exposed the masseteric fascia, the branches of the facial nerve, Stenson's duct, and a portion of the parotid gland. The deep fascia and masseter muscle are then divided by a transverse incision below and parallel to Stenson's duct, cutting directly down to the

bone about a centimetre below the sigmoid notch. Great care must be taken not to wound any of the lobules of the parotid whilst so doing, for even though the main duct be not divided a salivary fistula may ensue, leading to interference with healing of the wound. The outer surface of the jaw is next denuded of periosteum by means of raspatories, and the soft parts held aside by suitable retractors to allow of the application of a trephine, the diameter of which should not be less than $\frac{3}{4}$ in. It should be so applied as to leave between it and the sigmoid notch a narrow bridge of bone which can be subsequently clipped away by cutting pliers, and a sufficient amount of bone in front and behind to preserve the continuity of the jaw with the articular and coronoid processes. (Fig. 13.) At this stage the inferior dental artery may be cut through by the trephine and give rise to troublesome hæmorrhage. The disc of bone having been lifted out and the bridge of bone between the condyle and coronoid process clipped through with bone pliers so as to increase the space in which to work, some loose fatty tissue presents and should be carefully picked away with two pairs of dissecting forceps. The tendon of the temporal muscle is thus more clearly defined, and must be held forward, if necessary. Narrow spatulæ are useful at this stage, not only to keep the wound open, but also by their pressure to arrest hæmorrhage from divided muscular branches. If the bleeding is troublesome the wound should be packed for a few moments with small pieces of sponge wrung out of hot 1-40 carbolic lotion, any obvious bleeding point being secured by ligature. The inferior dental artery, if still intact, is usually first seen and may be secured by passing two ligatures around it with an aneurism needle and dividing it between. The lowest fibres of the external pterygoid muscle are seen running transversely across the wound and require to be held upward or carefully divided, to demonstrate the two nerves passing down from behind. The trunk of the inferior dental nerve can then be raised upon an aneurism needle, and the lingual found a little internal and in front; indeed it occasionally happens that the nerves lie in such close proximity to one another that they are picked up together. A silk or catgut ligature may be advantageously passed around them in order to be able to make traction. It is now easy by a little manipulation with the handle of a scalpel to trace them up to the foramen ovale, which can even be seen, if the external pterygoid muscle be held well

out of the way. The nerves can then be divided close to the skull either by scissors or knife, and the meningeal artery should be in no danger if the nerves have been sufficiently isolated. Peripheral traction is also employed so as to draw up as much of the nerve as is possible, and thus a considerable portion—more than an inch—of the trunks can be readily removed. All bleeding having been arrested, the wound is irrigated with some 1-40 carbolic lotion, and the skin flap neatly brought together with a continuous suture. There is no need to insert a drainage tube, and as a rule healing by first intention is readily obtained. I have performed this operation repeatedly during the last seven or eight years, and always with relief to the patient for the time being; but I must also confess that in by far the majority of cases the pain has recurred at the end of a year or two, necessitating those further measures which I shall detail in my next lecture.

LECTURE III.

The Gasserian Ganglion and its Removal.*

MR. PRESIDENT AND GENTLEMEN,—In my previous lectures I have traced the history and gradual development of the surgical treatment of trigeminal neuralgia, showing clearly the tendency amongst surgeons in this and other countries to substitute more central and radical measures in the place of less effectual peripheral treatment. For, however great the relief may be for a time after neurectomy, or combined neurectomy and nerve-stretching, a recurrence of the painful symptoms is unfortunately the rule rather than the exception. In a few months, or perhaps at the end of a year, these patients apply for further treatment, and in many instances are willing to undergo any risk from an operation that holds out the slightest prospect of alleviating their

* The term "Casserian" has also been applied to this ganglion, but incorrectly so, as it is named after Johann Laurentius Gasser, an anatomist of the 18th century, of whom nothing is known save that he was the instructor of Antonius Raymond Balthasar Hirsch, who in 1765 named the ganglion after his teacher. (New Sydenham Society's 'Lexicon of Medicine,' 1888.)

sufferings. They have told me that life under such circumstances is unendurable, and have even threatened self-destruction when the paroxysms have been extremely severe, reason itself being endangered; whilst others have expressed the hope that they might never regain consciousness from the anæsthetic administered at the operation, rather than again experience such exquisite agony. It was a case of this desperate character that first stimulated me to make an attempt to reach and remove the Gasserian ganglion; and after conversations with Dr. Ferrier and Mr. Horsley, and a careful study of the anatomical relations of the ganglion to the base of the skull, I was convinced not only of the possibility but also of the practicability of this proceeding. The results more or less favourable following the removal of Meckel's ganglion also acted as a powerful argument in favour of producing a still more satisfactory effect if the Gasserian could be reached and isolated from the brain, even if the whole ganglionic mass could not be excised.

Surgical Anatomy.—The *Gasserian* or *Semilunar ganglion* is a small mass of greyish-yellow nerve substance, analogous to the ganglia upon the posterior roots of the spinal nerves, as it forms an expansion upon the larger or sensory root of the trigeminal nerve, the motor root being separate. The trunk of the fifth nerve issues from the pons as a cord flattened horizontally, and passes through an opening in the dura mater to reach the apex of the petrous portion of the temporal bone where the ganglion is lodged. This opening is placed immediately behind the posterior clinoid process in the substance of the anterior attachment of the tentorium, and has the superior and inferior petrosal sinuses respectively above and below it. The space in which the ganglion is located is known as the *Cavum Meckelii*, and corresponds exactly to the depression at the apex of the petrous bone. It is essentially an extra-dural space, a matter of considerable importance in any operative interference. Some French anatomists, however, state that the space is lined below by a thin reflection of the dura mater, and thus the ganglion really lies encapsuled within the dura. In shape the ganglion is crescentic, or somewhat like a haricot bean flattened out, with the convexity forwards. The antero-external surface is in somewhat close relation with the dura mater, from which it is not easily separated; the postero-internal surface lies on the bone, a thin layer of dura, acting as

periosteum, intervening, and with this it is very loosely connected. The trunk of the fifth nerve enters its postero-superior border at a point corresponding to the hilum, and at the antero-inferior border are given off the three main divisions (Fig. 14). The

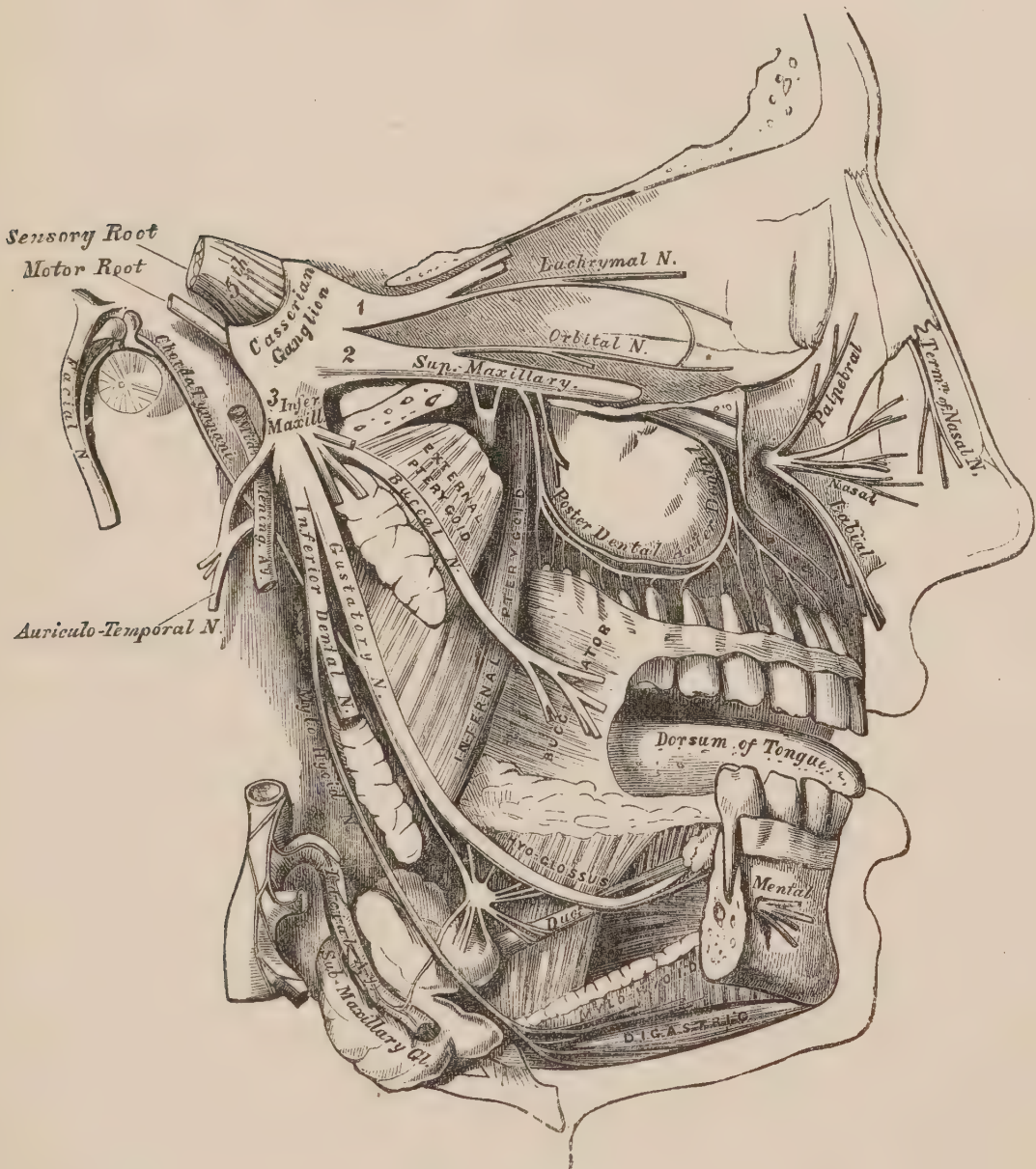


FIG. 14.—Gasserian ganglion and its branches. (Longmans & Co.)

anterior portion is prolonged into the ophthalmic division, which is very closely connected with the dura mater forming the outer wall of the cavernous sinus, and it is a question whether the removal of this portion is possible without laceration of the sinus.

Thiersch and Horsley both emphatically contend that the detachment of this portion is impossible; but recent investigations have convinced me that on the cadaver at least it can be accomplished, though not without some difficulty. The internal carotid artery passing upward in its canal in the petrous bone lies first below and then internal to the ganglion, previous to gaining its position by the side of the posterior clinoid process in the floor of the cavernous sinus.

Operative Treatment.—The various methods which have been suggested and practised of reaching the ganglion must now be considered. Up to the present this has been accomplished in three different ways, viz. :—

1. By Mr. Horsley's intracranial operation.
2. By ablation of the superior maxilla, and trephining the base of the skull; and
3. By trephining the base of the skull through the pterygoid region; this method, which is an elaboration of Krönlein's* modification of Pancoast's original proceeding, I have adopted in my last four cases.

I have recently received an interesting communication from Professor Andrews, of Chicago,† who, during the last twelve months, has been making investigations on the cadaver as to the best means of reaching the ganglion. He has also sent me details of two cases in which he has followed out my suggestions in his operation, and to which I shall presently refer. His studies have led him to much the same conclusions as to the best and most direct means of effecting this. He suggests six different routes; but, inasmuch as two of them are essentially very slight modifications of my own plan, they may, for practical purposes, be reduced to four, the three mentioned above, and a fourth which, however, is only suggested as possible on the cadaver, and is at once condemned by the author himself.

I. With regard to the *intracranial operation*, Mr. Horsley has so recently published his views on this subject,‡ and his method of operating, that a detailed description here will be unnecessary; briefly, it consists in the exposure of the squamous portion of the

* 'Deutsche Zeitschrift für Chirurg.,' xx, p. 484.

† Papers read in Section of Surgery and Anatomy at 'Amer. Surg. Ass.,' 1891, p. 153.

‡ 'British Medical Journal,' December 5th, 1891.

temporal bone by turning down a large temporal flap and in the removal of the whole of the exposed bone by means of a trephine and bone forceps. The dura is then freely divided along the whole length of this opening, and the temporo-sphenoidal lobe laid bare. "A broad copper retractor with smooth and everted edges is then gently slipped underneath the lobe, and slowly but steadily raised. The lobe is partly moulded, partly lifted upward, and the floor of the skull is then seen and illuminated by the electric light." The edge of the tentorium is looked for, and the position of the root of the nerve, as it emerges from the brain, ascertained. The ganglion will not be visible at present, being covered by the dura, but an incision is made at the point where he considers it should be, and by enlarging this the nerve root is stated to be seen with ease, "freely lying in the little canal," to use his own words, and can then be divided and the ganglion removed. The first case Mr. Horsley treated in this way proved fatal in seven hours from shock, and, as far as I can make out from the information given in his table of results, he had not, when his paper was written, attempted a similar operation again, though in two cases an intra-cranial division of the nerves was undertaken, the exact details of which are not stated, but in neither of these was the ganglion dealt with.

An examination of the inside of the base of the skull from which the brain has been recently removed is sufficient to convince one of the difficulties which must be experienced in attacking the ganglion from above; much more so is this the case with the brain *in situ*. Again, when we consider that the ganglion is situated practically outside the dura mater, it seems an unnecessary increase in the severity of the operation to open this investment in two different situations; and, however delicately we may handle the temporo-sphenoidal lobe, it can do it no good to compress and mould it by retractors to a sufficient extent to enable the base of the skull to be clearly seen, and the necessary manipulations undertaken, all of which entail a considerable degree of shock. Moreover, Mr. Horsley himself admits that several small veins passing from the temporo-sphenoidal lobe to the superior petrosal sinus must be torn in this proceeding, and so give rise to troublesome hæmorrhage, and the superior petrosal sinus itself will hardly escape, if an incision be made through the dura in the direction indicated. Bleeding in this locality is difficult to arrest,

and even if not absolutely dangerous to the patient from its extent, it may become so by its pressure effects. Again, the extensive removal of cranial bone must be a source of subsequent risk to the patient, and the disfigurement of the face from the large temporal flap is very marked. These latter points, however, would be of little importance if certain relief could be given to the painful condition without danger to life. From these considerations one cannot resist the conclusion that this method of reaching the ganglion is scarcely justifiable in the light of present experience, and that if it can be satisfactorily dealt with along some safer route, it is far better to avoid the risks certainly associated with such an extensive intra-cranial operation.

II. My own efforts hitherto have been entirely directed to reaching the ganglion through the base of the skull, and at present, after an experience of five cases, I see no reason for altering my views. In the first case, one condition on which I was allowed to operate was, that I should remove what was thought by the patient to be the seat of greatest pain, viz., the right superior maxilla; under no other circumstances should I have undertaken such a disfiguring plan of treatment. The operation of *ablation of the superior maxilla* was performed in the usual way without any difficulty, and, as generally happens, the pterygoid processes were broken off close to their base. This gave me plenty of room to expose the foramen ovale, and the trephine was easily applied in a direction upward and a little backward. After the removal of the disc of bone containing the foramen as its centre, the ganglion could be easily seen both by myself and my assistants by the aid of the electric illuminator, without which manipulations at such a depth would be impossible. At that time I was not provided with the special hooks I have used in my later cases, and so I picked the ganglion away piecemeal with a pair of fine-hooked forceps. As the wound communicated with the mouth, its asepticity could not be maintained. This condition may account in part for the loss of the eye, but with this exception the patient made an excellent recovery. I regret that I did not take proper precautions to render the conjunctival sac aseptic before the operation, or to protect the eye subsequently. It is possible, moreover, that a drop or two of chloroform may have fallen into and irritated it, as the patient was somewhat restless. Be that as it may, subsequent experience would tend to prove that, unless

the strictest precautions be taken for the protection of the eye at the time of the operation and for weeks after, the disturbance of the trophic centres may lead to degenerative changes entailing the loss of the organ. But even if the eye should unfortunately be lost, the immunity from pain more than compensates for this.

III. My present operation, *through the pterygoid region*, must be described in detail.

Preparation of Patient.—The patient should be in as favourable a condition as possible. The bowels are moved by a mild purgative given the night before. The face is washed as thoroughly as the patient will permit with soap and carbolic solution (1-20) some hours before, and an antiseptic dressing applied. This is important, seeing that (as I have previously remarked) the skin is often very dirty on account of the pain caused by any attempts at washing. Chloroform is, perhaps, the most convenient anæsthetic to employ, and when the patient is fully under its influence, the skin and external ear should be again thoroughly cleansed, and a plug of salicylic or some antiseptic wool inserted into the meatus. The conjunctival sac must also be thoroughly washed out with an efficient but unirritating antiseptic, a 1-2000 solution of corrosive sublimate being, perhaps, the most satisfactory; during this proceeding the lachrymal sac should be squeezed, as collections of mucus are often found therein, and regurgitation of these through the canaliculi may cause septic contamination of the conjunctiva. In order to ensure closure and protection of the eye, both during the operation and for some days after, two fine horsehair or catgut sutures are introduced through the integument of the upper and lower lids, about 2 mm. from the palpebral margin of either lid, and exactly opposite each other, taking up small folds of the lax skin, which are approximated by tying the sutures.

The operation itself may be divided into six stages, as follows:—

1. Incision through skin and reflection of flap.
2. Section of zygoma and coronoid processes, and detachment of masseter and temporal muscles.
3. Exposure of the base of the skull, and search for the foramen ovale.
4. Opening the base of the skull.
5. Removal of ganglion.
6. Reposition of displaced structures, and closure of wound.

STAGE I.

The skin incision (Fig. 9, c) is made by entering the knife over the malar bone about half-an-inch below the external angular process of the frontal, and carrying it along the zygoma, and down in front of the ear over the parotid region to the angle of the jaw, and then forward along the lower border of the horizontal ramus as far as the facial artery. This done, a flap can now be dissected forward, consisting of skin and subcutaneous fat only, care being taken not to injure Stenson's duct or any of the branches of the facial nerve which lie in close contiguity to the masseteric fascia. Before this is completed, either a fine catgut thread can be inserted in either side to form a landmark in the subsequent suturing of the wound, or a cross-cut can be made in the skin. The scar resulting from this incision can be rendered almost imperceptible if great care be taken in stitching the parts together and accurately matching them up.

Many other incisions have been devised for similar operations in this neighbourhood, but they are not so satisfactory. Krönlein used an incision which was practically H-shaped, the transverse piece being placed over the zygoma. Pancoast operated through vertical incisions placed along the anterior and posterior borders of the masseter muscle and joined by a transverse cut along the zygoma. The great objection to these is the anterior vertical incision, which is very disfiguring and of doubtful utility. It is perfectly feasible to dissect up a skin flap from the face without encroaching on the nerves, and I would contend that the curved incision, whilst it gives a maximum of space with a minimum of disfigurement, will in no way interfere with the subsequent mobility of the facial muscles.

The skin flap must be carefully protected by a few layers of purified gauze during the operation, and not unnecessarily handled, or exposed to pressure or rough manipulation. It is better to avoid retractors in order to hold it out of the way, and its temporary fixation by a catgut suture to the upper part of the chin will be found beneficial. The anæsthetist should prevent any chloroform dropping on the under surface of the flap, and not allow any part of his apparatus to touch it. Particular stress is laid upon these details, as it is most important not only to maintain asepsis and obtain primary union, but also to leave as little

trace of the surgeon's work as possible. After raising the flap any arterial hæmorrhage of importance should be controlled by Spencer Wells' forceps, and probably the transverse facial vessels will need ligature.

STAGE II.

Section of Zygoma and Coronoid Process, and Detachment of Masseter and Temporal Muscles.—The zygoma is now exposed by means of suitable raspatories and periosteum detachers through an incision along its course. Two holes are drilled at the root of the zygoma, and two also anteriorly through the zygomatic process of the malar bone. This is best accomplished by a fine drill driven by a dynamo. The drill used should be of such a size that the perforation in the bone may carry wire of gauge No. 22, and the holes should be about one-third of an inch apart. The bone is then divided between them with a fine saw, and in such a way that the anterior saw-cut is directed obliquely downward and forward, the posterior part of the zygoma being divided as near

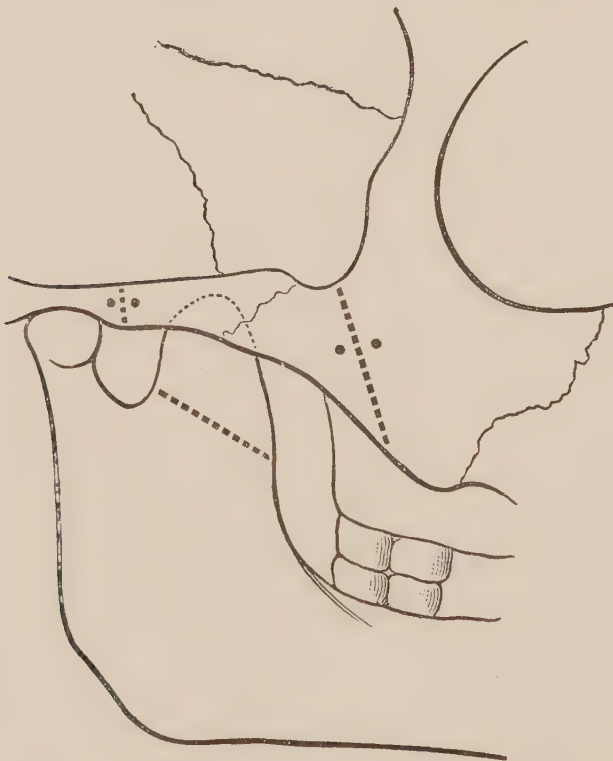


FIG. 15.—Diagram of zygoma and lower jaw *in sit u*. The interrupted line represent the direction of the saw cuts, and the dots on either side of those in the zygoma the drill holes.

its root as possible (Fig. 15). It is obvious that the bone can be drilled much more efficiently whilst the zygomatic arch is intact, and can be subsequently replaced without difficulty, and maintained in position by means of silver wire.

The zygoma thus detached is displaced downward and backward together with the masseter; to facilitate this it will be necessary to completely divide the muscular fibres attached anteriorly to the malar bone. Necrosis of the zygoma has occurred in some instances where it has been detached and turned down by this plan of treatment; but such was probably due either to septic contamination of the wound, or to some rough handling of the bone which might have been avoided. The attachment of the fibres of the masseter muscle to its under surface, from which its nutrition is derived, must not be interfered with; and in all probability the wiring of the bone into position after preliminary drilling is another preventive of necrosis, for in none of my own cases have I had the slightest trouble or subsequent difficulty. When the masseter has been sufficiently depressed consistent with the integrity of the important adjacent structures, and a little cellular tissue picked away, the coronoid process will be exposed together with the tendon of the temporal muscle, which passes further down on the inner aspect of the bone than on the outer. In Cases II, III, and IV this process was drilled to provide holes for subsequent wiring, and then divided obliquely downward and forward (Fig. 15). The detached bone was turned up with the temporal muscle, and the deep fibres encroaching upon the ramus of the jaw carefully divided. But latterly I have questioned very much the advisability of attempting to gain osseous union of this process; for the temporal muscle attached to it is paralysed by the operation, and necessarily atrophies, with the result that very considerable impairment of the mobility of the jaw ensues. In my last case, therefore, I simply divided the coronoid without attempting to drill it, and drew it and the muscle upward out of the way, subsequently excising it and a portion of the tendon.

STAGE III.

Search for the Foramen Ovale.—A certain amount of loose cellular tissue and fat will now present, under which will be found the external pterygoid muscle, running transversely backwards, to

be inserted into the condyle of the jaw (Fig. 16), and perhaps below it a small portion of the internal pterygoid may be seen. Running superficially across the former muscle, between it and the jaw, the internal maxillary artery is usually found, passing into

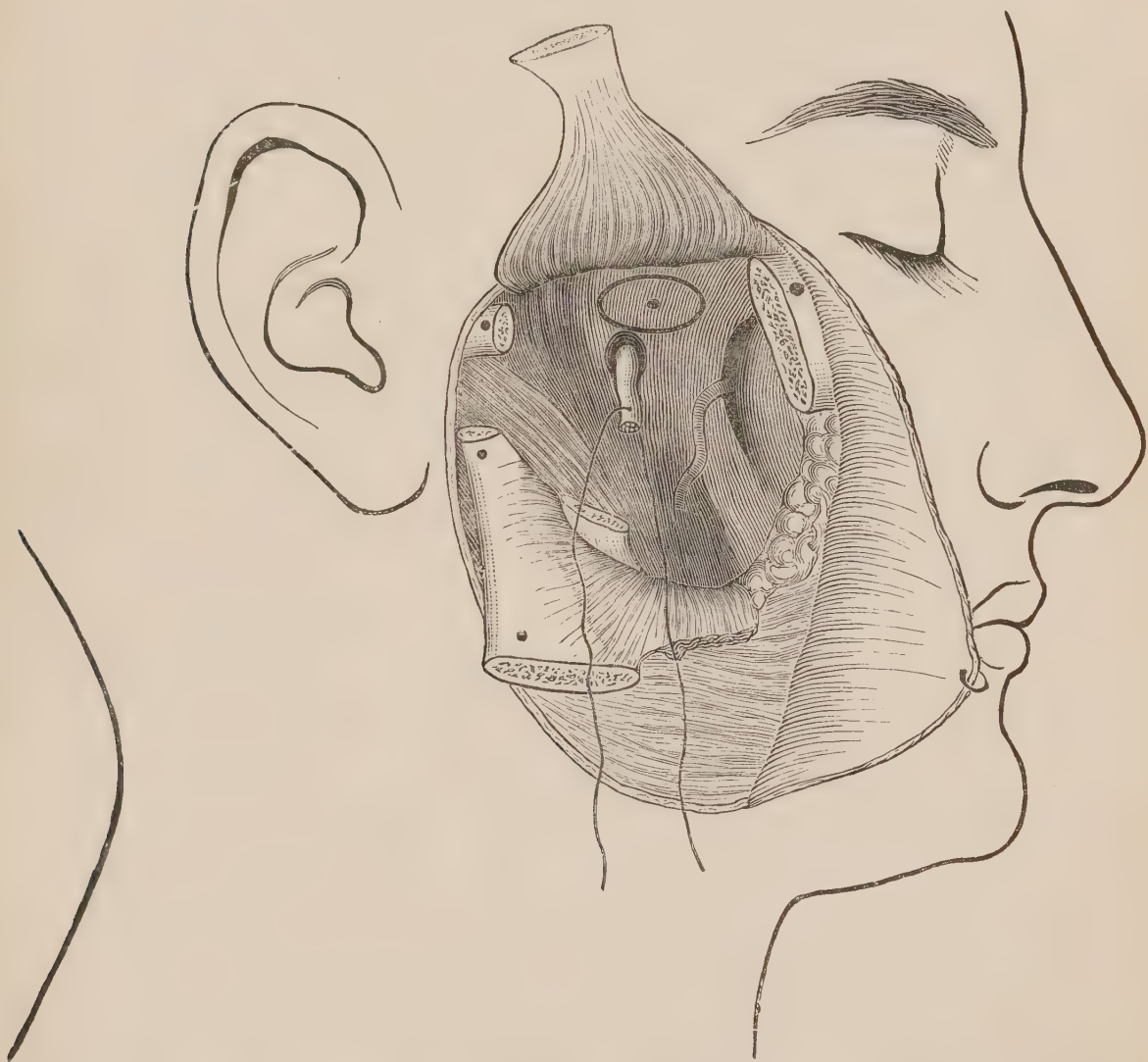


FIG. 16.—Sketch from dissected subject, showing stages of operation. The zygoma and masseter are turned downward and backward; the coronoid and temporal upward; the great wing of the sphenoid exposed by detachment of external pterygoid, showing relative position of foramen ovale and trephine track. (For clearness sake the two latter are purposely placed a little forward.)

the speno-maxillary fossa between the heads of the muscle. The artery, if it has not been tied at a previous operation, should now be sought for, and divided between a double ligature; by this

means hæmorrhage, which might be troublesome during the later steps of the operation, will be avoided. The inferior dental and gustatory nerves under normal circumstances pass downwards from under the lower border of the external pterygoid muscle; but if they have been previously removed, their assistance in guiding the surgeon to the foramen ovale is not available. The external pterygoid muscle is next detached from the great wing of the sphenoid and from the outer surface of the external pterygoid plate by scraping it from the bone with suitable raspatories from above downward. The knife should be used as sparingly as possible, and the strands of muscular tissue are best picked and cut away with dissecting forceps and a fine pair of blunt-pointed scissors. By this means the under surface of the great wing of the sphenoid is exposed, as well as the outer pterygoid plate.

The foramen ovale is now to be brought into view, a matter often of some difficulty, and the occasional reference to a dry skull held by an assistant will be a considerable help in indicating its position in relation to the neighbouring landmarks. It is usually on a level with the eminentia articularis, but occasionally lies a little behind it. In fact, the portion of bone which one first reaches in this deep part of the operation is well in front of the foramen, and one is apt to get too far forward, so that the pterygo-maxillary fissure is mistaken for it. In the third of my cases this actually occurred, and at first I trephined the sides of the fissure, not discovering my mistake until I found orbital fat protruding from the opening. The relation to the root of the pterygoid processes is another guide; the foramen lies usually a little behind and external to the base of the outer plate, but sometimes directly behind it. The position, however, is not constant, and Mr. Carless has found the greatest variety in the skulls which he has examined for me. The base of the external pterygoid plate, he states, is by no means a fixed guide, in that in many old skulls there is a formation of bone (like a tongue) projecting backwards towards the spine of the sphenoid, usually *external* to the foramen ovale, and deeply channelled or grooved for the middle meningeal artery. M. Testût* also mentions this fact, stating that it is due to an ossification of the pterygo-spinous ligament of Civinini. The sphenoidal spine lies immediately behind the foramen ovale and about a centimetre from it, the foramen spinosum intervening and

* Testût, 'Traité d'Anatomie Humaine,' 1889, vol. 1, p. 113.

placed about 2 mm. behind the oval opening; the spine cannot, however, always be felt on the living subject on account of the depth of the wound and the limited space in which one is working. Under these circumstances, it is important to define clearly with the finger the outer pterygoid plate, and help may be obtained from the facts ascertained by the measurement of a number of skulls by my colleague, that in an adult male skull the average distance from the anterior border of the outer pterygoid plate (*i.e.*, from the posterior lip of the pterygo-maxillary fissure) to the centre of the foramen ovale is about 18 mm., whilst in the female adult skull it is about 16.5 mm.; in both sexes, the average measurements are a little greater on the right side; but in skulls that are abnormally large or small they vary to a corresponding degree. Should the above-mentioned pterygo-spinous ridge of bone be met with it may be necessary to clip it carefully away, in order to define the position of the foramen.

STAGE IV.

Opening the Base of the Skull.—Having exposed the foramen ovale and traced to it either the trunk of the undivided lingual and dental nerves, or the stump left from former operations, the base of the skull is now to be opened by means of the trephine. My intention in Cases 2, 3, and 4 was to remove a disc of bone having the foramen ovale for its centre, and for this purpose I employed a trephine with a handle set on a stem long enough to clear the cheek, and with a reversible centre pin, one end of which was pointed as usual, the other blunt. The size of the trephine was such that it should remove a $\frac{1}{2}$ -inch disc of bone; the crown was serrated obliquely on the outer side for a distance of a quarter of an inch, in order that the trephine might clear itself of *débris*, and not get jammed in the bone. This trephine is similar to one used by Mr. Horsley, with the exception of the reversible centre pin.

The smooth-ended centre pin was projected as far as possible, and passed into the foramen ovale, so that the blunt point might protect the dura mater or other intra-cranial structures by pushing them before it. To do so, the trephine was, roughly speaking, held in such a position that its axis was parallel to the external pterygoid plate. The handle of the trephine was then depressed

and kept as far back as possible ; but from the pressure of the soft parts it was always applied at an angle, and not perpendicular to the surface, a proceeding not altogether undesirable, inasmuch as thereby the integrity of the carotid canal could be more readily maintained. The close contiguity of this structure had to be carefully kept in view during this stage of the operation, and, indeed, there is only the inner border of the great wing of the sphenoid, measuring from 2 to 4 mm. in thickness, intervening between it and the foramen ovale. By holding the trephine at this angle, the outer segment of the bony circle was cut through first ; the bone could then be broken off on the inner side along the sutural line between the apex of the petrous bone and the great wing of the sphenoid, and thus the carotid canal remained uninjured. But even if it should be encroached upon, it by no means follows that the artery lying within will be damaged, as there is always a certain amount of space to allow of its expansion and other movements under the blood pressure. The disc of bone being now set free by an elevator, will fit like a collar over the stump of the divided nerve, and can be slipped over it. In one or two of my cases I noticed a definite constriction of the nerve at this point.

In Case 4 I made an important modification by trephining in addition the great wing of the sphenoid anterior and a little external to the foramen. The trephine openings were then united by removing the intervening bridge of bone by a careful use of chisel and mallet. During this process the dura mater, which had been previously loosened around the openings, was protected by a copper spatula, and held up out of the way, for, when unsupported, it bulged through the opening. As will be seen by reference to the account of my cases which I append, this patient did well, but during the first forty-eight hours after the operation she had a certain amount of epistaxis, and also vomited some grumous material, like coffee-grounds, which was evidently altered blood. The source of this was a matter of considerable anxiety to me ; it evidently did not come from either the sphenoidal sinus or the antrum, as these were in no way interfered with, but, on careful examination of the base of a skull, the relationship of the Eustachian tube, which had been previously overlooked, seemed clearly to indicate whence the bleeding was derived. This structure lies in immediate contiguity to the ridge of bone which forms the

inner boundary both of the foramen ovale and spinosum. A groove will be found in this position in most skulls extending backward to the point of attachment of the tube to the petrous portion of the temporal bone and forward to the base of the pterygoid process; and this depression is occupied by the cartilaginous portion of the tube. It is highly probable that in removing a disc of

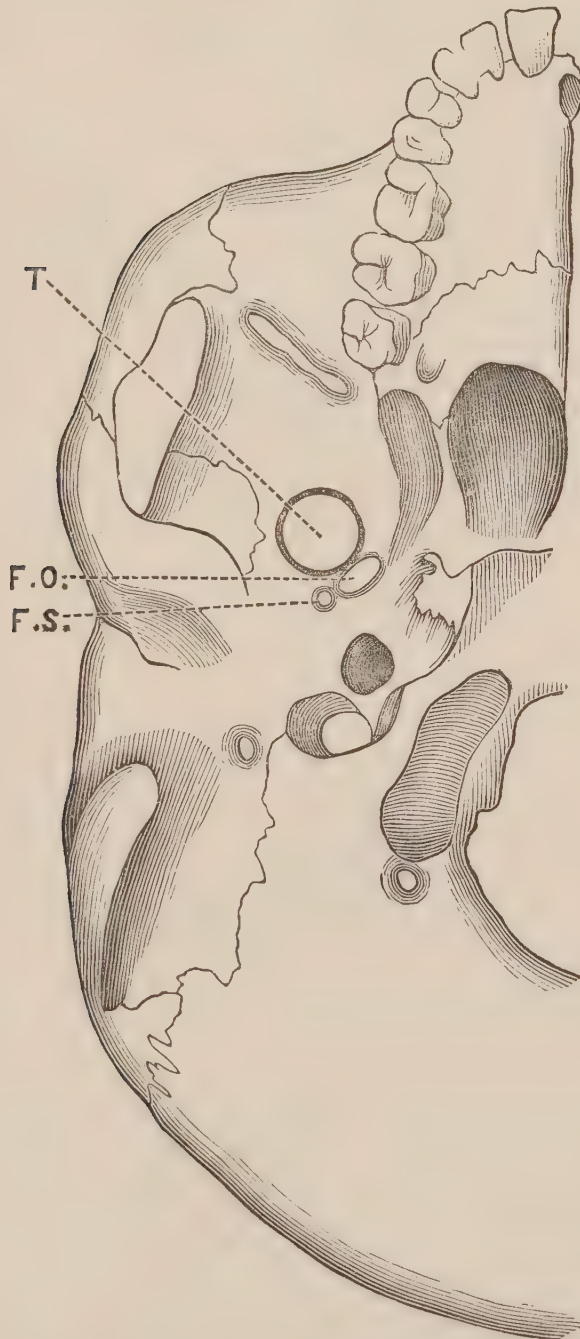


FIG. 17.—Diagram of base of skull, showing position of trephine track.
T, Trephine track; F O, Foramen ovale; F S, Foramen spinosum.

bone, half an inch in diameter, with the foramen ovale as its centre, this structure will be encroached upon, laying the wound open to the risk of septic contamination from the pharynx. This consideration was one of several, which induced me to alter my plan of operation in the last case I dealt with; I applied the trephine to the great wing of the sphenoid a little anterior and external to the foramen, and in such a way that the circumference of the disc just impinged on its outer wall (Figs. 17 and 18). The

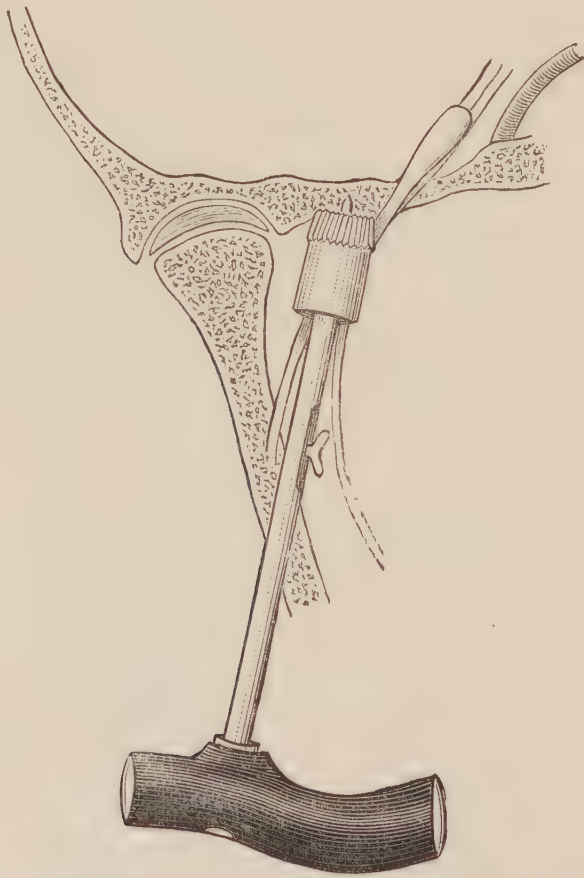


FIG. 18.—Diagrammatic section of base of skull and lower jaw showing relation of trephine to foramen ovale and Gasserian ganglion. (After *Andrews*.)

opening thus made can be subsequently enlarged, if necessary, in any direction desirable. It must not be forgotten that the thickness of the skull is very unequal, being thinner on the outer margin of the trephine track than on the inner; and inasmuch as the instrument is necessarily applied at an angle, the outer half will be cut through before the inner. This fact renders damage to the dura possible in spite of the most careful precautions.

STAGE V.

Removal of the Ganglion.—Having repressed the prominent dura with a spatula, the trunk of the third division, which during all these preliminary proceedings has been carefully guarded, and round which a ligature should now be passed, is to be traced up to the ganglion, which should be loosened from its resting place upon the apex of the petrous portion of the temporal bone. No great difficulty need be experienced as regards the posterior half, but

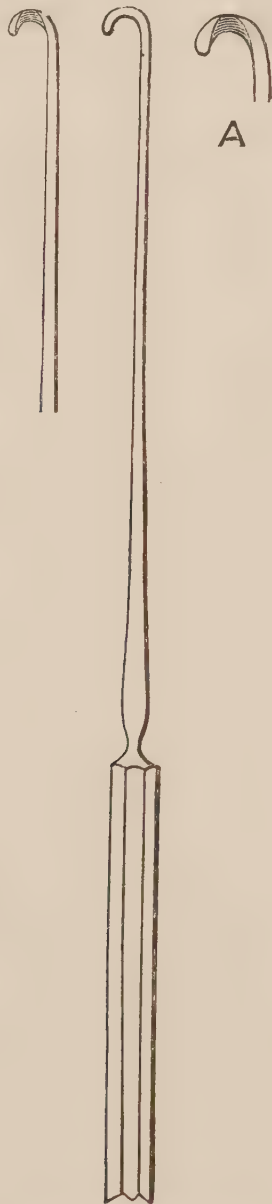


FIG. 19.—Hooks for dealing with the ganglion (actual size). A, Enlarged sketch of hook, to show concave cutting edge.

inasmuch as the anterior and upper portion is closely incorporated with the dural sheath, it is perhaps better to sever the connection between the ganglion and the brain at its exit from the dura, and then to draw it forward with a delicate pair of forceps. For this work a pair of fine hooks, such as those made for me by Mr. Hawksley (Fig. 19), will be found most useful; one of them is an ordinary blunt-pointed hook to pass round the nerves and free them from their connections; the other has a sharp edge upon its concave aspect to be used for cutting them through. A pair of long-handled strabismus scissors may also be required. It is quite possible that in dividing the root of the nerve just outside the dura a prolongation of the subdural space may be opened, and a small quantity of cerebro-spinal fluid escape through it; this, however, will be of slight extent, and is of no moment, if the wound be kept aseptic. The second division of the nerve must now be dealt with, and this may be facilitated by enlarging the opening in the base of the skull in the direction of the pterygoid processes, and holding up the dural wall out of the way; it may be divided just in front of the ganglion by the sharp hook. Having thus severed all its connections, except the ophthalmic division, the ganglionic tissue, which is exceedingly soft, may be pulled away piecemeal by forceps or by a small curette as recommended by Professor Andrews. In this way the danger of wounding the cavernous sinus is reduced to a minimum.

STAGE VI.

Reposition of Displaced Structures and Closure of Wound.—After the steps detailed above have been satisfactorily accomplished, the toilette of the wound must be attended to. The bleeding having been staunched, the parts should be thoroughly washed with a warm 1-40 solution of carbolic acid. The coronoid process may be either sutured or removed; in my last case I removed it. Silver wire should be passed through the holes previously drilled in the zygoma, so that it can be accurately adjusted. By this means the contour of the face will not be interfered with, the chances of necrosis or collapse of the zygoma prevented, and firm bony union assured. The integuments may now be brought together with a continuous fine catgut suture, care being taken to adapt the parts accurately to each other. No drain tube is needed, although in

my first two cases I used one as a precautionary measure. If the wound has been occasionally irrigated during the operation, and complete asepsis maintained, primary union may certainly be expected. I am glad to say that no suppuration has occurred in any of my cases. To prevent accumulation of blood in the wound, gentle but continuous pressure by means of a purified sponge introduced between the second and third layers of the cyanide gauze will be found very efficacious for the first forty-eight hours. In addition to the face dressing, both eyes should be carefully covered with pads of salicylic wool, and lightly bandaged. A certain amount of shock necessarily follows such a protracted operation, and a subsequent elevation of temperature must be expected at the end of the second day. Beyond this no symptoms of importance have been exhibited. The dressing has usually required changing once or twice in the first four days, at the expiration of which time it may be replaced by gauze fixed down with collodion. The stitches can be removed at the end of a week, if they have not already been absorbed. The eye should be kept closed for at least four days, when the stitch in the lids may be removed; but it is safer to keep both eyes bandaged for a week, and the eye on the side operated on for three or four weeks.

I now append a brief *résumé* of my cases:—

CASE 1.—Mrs. F. M., aged 60, sent to me by Dr. Padman, of Bloomsbury, first came under my care in 1888. She had been suffering for five years from severe neuralgia, which had specially affected the inferior dental nerve of the right side. Counter-irritation and constitutional treatment having failed, the dental nerve was stretched at the entrance to its canal through the mouth on August 19th, 1888, and at the same time was divided at its exit from the mental foramen. This was followed by relief until March, 1889, when the pain returned with great severity, for which the lower jaw was trephined and half an inch of the nerve excised. Partial relief followed for a year; but in March, 1890, the lingual and dental nerves were cut down upon by deepening the sigmoid notch, and divided close to the base of the skull. The pain now recurred in the second division with greater intensity than before, any touch upon the upper jaw of that side producing a shock of agony terrible to witness. On April 2nd, 1890, I performed ablation of the superior maxilla, and trephined around the foramen ovale for removal of the ganglion (p. 210). The patient made an excellent recovery, with the exception of the ocular trouble already alluded to. The latest report, dated January 28th, 1892, states that she is still quite free from pain, and her general health very good.

CASE 2.—Mrs. S. C., aged 63. The neuralgia had commenced ten years before she came under observation, in connection with the teeth of the

right lower jaw, and, in spite of all treatment, it spread to other branches and increased in severity. In 1884 all the teeth on the right side both of the upper and lower jaws were extracted, but without any relief. Drugs and local applications had no effect. When first seen by Dr. Ferrier and myself, the case was typical, a paroxysm being elicited by almost any movement of the jaw or stimulus from without, the pain chiefly affecting the lingual and dental nerves, and the back of the orbit, and shooting down into the palate. In consequence of the evident implication of the second as well as the third division of the trigeminal, Dr. Ferrier recommended that the Gasserian ganglion should be dealt with at once, without having recourse to any minor measures. As I entirely agreed with this opinion, the operation was undertaken on January 29th, 1891. The foramen ovale having been exposed through the pterygoid region was trephined, and the opening increased by a larger trephine. The ganglion was then removed piecemeal. The patient made an uninterrupted recovery. The cornea was anæsthetic when exposed four days after the operation, and the conjunctival reflex was diminished for some time, but no trophic disturbance manifested itself. Slight difficulty in mastication was subsequently experienced, but this soon passed off. There has been no recurrence of the neuralgic pain, and the patient was seen by me only a few days ago (*i.e.*, about the end of January, 1892).

CASE 3.—Mrs. E. K., aged 63. The neuralgia in this patient had lasted for about five years, having originated in the inferior dental nerve as the result of carious teeth, which were removed, but without benefit. In February, 1890, I divided the inferior dental and gustatory nerves close to the foramen ovale by deepening the sigmoid notch, and excised about half an inch of each. This was followed by relief for about a year, when she was re-admitted to the hospital with pain in the second and third divisions, but especially referable to the former. The operation for extirpation of the Gasserian ganglion was performed on October 29th, 1891, by the usual incision and with the usual precautions. The great wing of the sphenoid and external pterygoid plate having been cleared, what was considered to be the foramen ovale was exposed and the trephine applied; the appearance of some orbital fat, however, through the trephine hole, indicated that the pterygo-maxillary fissure had been mistaken for it. The raspatory was again employed further back, and the foramen ovale clearly demonstrated. Owing to the previous operation there was less hæmorrhage than usual, but the tissues were much matted together, and so interfered with the application of the trephine. The base of the skull was opened, and as much of the ganglion as could be seen removed. The wound was treated as usual, and healed without trouble, both the coronoid process and zygoma being wired. For a time the right eye was painful and the conjunctiva congested with some œdema of the lids, but there was no disturbance of the corneal epithelium. The greater portion of the right side of the face was anæsthetic for the first five days after the operation, but this gradually diminished in extent, so that when she left the hospital, on November 30th, there was a partial return of sensation over the first and third divisions. The cheek, lower eyelid, and upper lip had, however, completely lost their sensibility to touch and pain, and the right side of the tongue was still dull. The patient returned a few days ago, complaining of inability to open the mouth, which I remedied by forcible depression of the lower jaw under an anæsthetic, and the use of graduated wedges subsequently. The only

other complaint was slight occasional pain and intolerance of light in the right eye.

CASE 4.—Mrs. D——, aged 37. In this patient, who is of a highly neurotic temperament, the disease seems to have originated about twelve years ago in a bad tooth in the right side of the lower jaw. In 1887, I trephined the lower jaw and removed half an inch of the inferior dental nerve, with temporary relief, but the pain recurred under the right eye, in the tongue, and again along the lower jaw. In July, 1890, portions of the inferior dental and gustatory nerves were removed by deepening the sigmoid notch, but the relief given by this proceeding was of short duration, the pain recurring with more than its old severity, and extending down into the neck and up to the temple. On November 5th, 1891, I operated for the removal of the Gasserian ganglion. The operation was performed in the way detailed above, and the skull trephined in two places, one posteriorly having the foramen ovale for its centre, and the other a little anterior and external to it, the intervening bridge of bone being removed by chisel and mallet. In consequence of the thickened state of the 3rd division, it was difficult to draw the disc of bone over the stump, and when this had been accomplished, the foramen was found to be very small. By detaching the dura, the ganglion was seen and even felt by the finger, and by cutting through the different divisions was removed; the opening in the dura for the passage of the root of the nerve was clearly defined, and through this a small quantity of cerebro-spinal fluid trickled. During the removal of the ganglion, the dura which had a tendency to bulge, was lifted up by a bent spatula. The subsequent history of this case has been satisfactory up to the present. I have alluded already to the fact that some epistaxis and vomiting of altered blood occurred, probably from damage to the Eustachian tube. The wound healed throughout by first intention. One week after the operation the sensation of the right side of the face and tongue was carefully tested, and found to be much decreased, although not totally absent. Two or three weeks later the right eye became somewhat congested and irritable from the development of a crop of small subepithelial vesicles on the lower fifth of the cornea, which burst in a few days and left a superficial ulcer, which, however, readily healed under suitable treatment, and the congestion gradually disappeared. The eye pad was in this case retained for six weeks. The patient was examined on January 27th, 1892, and there had been no recurrence of the pain.*

CASE 5.—Mrs. B——, aged 37, sent up from Derby Infirmary by Dr. Taylor, had suffered from epileptiform tic of the 2nd and 3rd divisions for seven years. She had teeth extracted from both upper and lower jaws on the affected side, but this rather increased the trouble than otherwise, and the usual medical treatment had been tried without benefit. Paroxysms were excited by masticatory movements, even after removal of the teeth; they lasted about two minutes and recurred about every half hour. They could also be elicited by pressure over the points of exit of the 2nd and 3rd divisions. After consultation with Dr. Ferrier, and in consequence of the evident implication of both the supra- and infra-maxillary divisions, it was decided to cut down upon the Gasserian ganglion.

* But *vide* Addendum, p. 230.

January 16th, 1892.—The usual incision was made, and the zygoma, which in this case was very slender, was exposed, drilled, sawn through, and turned down. The anterior saw cut was made close to the malar bone, which was drilled on the slant to clear the root. The coronoid process was cut through, and the temporal muscle turned up and held out of the way. Troublesome bleeding from the pterygoid plexus of veins ensued, interfering for some time with the identification of the trunk of the internal maxillary artery, which was, however, finally secured. The external pterygoid muscle was scraped away from the skull, exposing the great wing of sphenoid together with the anterior margin of foramen ovale. The trunk of the nerve emerging from this was now isolated, ligatured, and divided below. The trephine was applied to the great wing of the sphenoid anterior and external to the foramen ovale, the circumference of the trephine track just touching it. Owing to the thinness of the bone in this position the edge of the trephine wounded the dura mater, so that the temporo-sphenoidal lobe was exposed. The trunk of the third division was lifted from the foramen into the trephine hole and traced up to the ganglion, which was then loosened from its connections, and probably the posterior half only removed. Before closing the wound, the coronoid process and about an inch of the temporal tendon were excised. The patient was somewhat collapsed after the operation, and complained of pains in the top of the head and in the limbs. The temperature rose to 101.4° on the second day, but subsequently remained normal. There has been none of the old pain since the operation. I shall anxiously watch the future progress of this case, for I think it highly probable that the anterior half of the ganglion was left completely undisturbed, and the cerebral root but partially divided, an occurrence attributable to the limited opening I made in the skull, to the wound of the dura, and to the troublesome hæmorrhage protracting the operation. The anæsthetic area in this case is not so complete as in the others, and seems mainly confined to the 3rd division. The sense of taste is completely lost on the right side of the tongue; this was carefully tested by Dr. Ferrier a few days after the operation with salt, sugar, and quinine.

A sixth case was operated upon in Chicago on November 7th, 1891, by Professor Andrews in a similar manner. The patient was a woman, aged 50, who had suffered for five years from "frightful pain along the inferior maxillary division without being able to obtain any relief, the act of swallowing being especially associated with the paroxysms." He trephined the base of the skull by the side of the foramen ovale, and broke up the ganglion thoroughly with a curette. The patient, who was very weak, felt the shock considerably, but rallied after a few hours, and was free from neuralgic pains. There was complete benumbing of all the parts supplied by the ganglion, and swallowing was no longer painful. The motor oculi nerve was paralysed, showing that it had been injured, probably by the curette. In ten days she was able to return to her home, 50 miles distant, although the wound was not quite healed, and it suppurated slightly afterwards. I had another communication from Professor Andrews recently, dated January 16th, 1892, stating that this patient had remained free from pain; that the paralysis of the motor oculi proved only temporary, but there was a slight tendency to pericorneal ulceration of the right eye.

He also sends me particulars of another case in a woman, 62 years of

age, the disease being on the right side, and involving the 2nd and 3rd divisions. He had previously removed the inferior dental branch close to the foramen ovale. The operation was performed in the middle of last December in the same way, by trephining just external to the foramen ovale, and removing the ganglion with a sharp curette. There was troublesome hæmorrhage during the operation. Up to the time of his writing the patient was perfectly free from pain.

RESULTS OF OPERATION.

In considering the results of partial or complete removal of the Gasserian ganglion, the first question which has to be answered is naturally, "What effect has this proceeding upon the pain?" Up to the present time we are able to give a satisfactory reply; all the five patients whom I have treated in this way have remained free from the typical and terrible paroxysmal attacks from which they had previously suffered. It is true that my first case was done only twenty-two months ago, and the last only sixteen days; consequently, it is too early to speak with confidence as to the permanent character of the relief: but the results hitherto obtained are sufficiently encouraging to lead me to continue in the same line of action. Absolute immunity from any kind of pain can hardly be expected after such a considerable disturbance of the structures at the base of the skull, and for some time there may persist a sore and stiff sensation in the region operated on and probably some wandering pains about the head; these are not considered of any moment by patients who have previously suffered such intense agony. The interference with the movements of the lower jaw is undoubtedly inconvenient, and renders the process of mastication a little difficult; but this may be avoided in the future by the removal of the coronoid process.

As to the effect upon the distribution of the sensory fibres of the 5th nerve, it is interesting both from the clinical and physiological sides to observe the rapid diminution of the anæsthetic area, and it would appear that sensation is re-established by the neighbouring branches much in the same way as collateral anastomosis takes place in the vascular system. This phenomenon is a fact which cannot be disregarded prognostically, although it is not necessarily the precursor of a relapse. The appearance of the side of the face operated on is characteristic of trophic disturbance; the skin has a shiny, somewhat injected look, whilst the hollows in the tem-

poral, pterygoid, and maxillary regions on that side clearly demonstrate the existence of muscular atrophy and cicatricial contraction.

The effect upon the nutrition of the eyeball is decidedly serious. In the first case, as previously mentioned, the organ was lost from suppurative panophthalmitis, and in two of the other cases the nutritive state of the globe was, for the time, considerably depressed. It is probable that the trophic centres are contained in the upper and anterior segment of the ganglion, and if this be so, the chances of damaging the eye may be lessened by leaving that portion intact, even though the trunk of the nerve be divided behind the ganglion. On the other hand, the interference with one part of the ganglion may induce degenerative changes in the remainder which will effectually prevent a recurrence of the malady, and yet will not be sufficient to cause permanent damage to the eye.

Such are the results, Mr. President and gentlemen, both of my own observations and experience together with that of others in this department of surgery, and from them I venture to draw two very definite conclusions: (1) that in severe cases of epileptiform neuralgia, both medical and surgical treatment have hitherto been unavailing to give permanent relief; and (2) that extirpation of the Gasserian ganglion through the base of the skull, though admittedly difficult, need not endanger life, and at present holds out the best prospect of dealing with these intractable forms of trigeminal tic. The test of time and further experience can alone decide what value will be finally attached to this measure; but at any rate the outlook is hopeful, and let us trust the future will not belie our present expectations.

In conclusion, once more let me thank you for the honour you have done me in electing me to this position, and for the patient hearing you have granted me.

APPENDIX.

Since the above lectures were delivered, I have operated on a sixth case with, I regret to state, a fatal result. The following are the notes of the case:—

Mrs. S—, a widow, aged 68, but looking much older, was admitted to King's College Hospital, on February 19th, 1892. About five years previously she was seized with acute pain in the skin over the right mastoid

process, and the adjoining part of the neck. It was noticed particularly on washing, and was confined to this area for about a year; it was paroxysmal in character, the attacks being only occasional and lasting two or three minutes. Two years later the whole of the right side of the face became affected; the pain appeared to start in the skin in front of the angle of the jaw, radiating along the ramus and into the infra-orbital region. The paroxysms became intensified in duration and frequency, and the patient was confined to bed during the winter of 1888-89 for ten weeks. In spite of treatment at Leicester and Buxton by drugs, baths, and electricity, in addition to which all her teeth were removed, the pains were increased in severity and extent, affecting the other side of the face and forehead, and the attacks became almost constant. On one or two occasions slight improvement seemed to follow the treatment, but such was only temporary. "Life had been miserable for the last year or two."

On admission, the patient was evidently in great pain, the paroxysms recurring with great frequency, being elicited by draughts or sudden movements of the head, neck or jaw, and also by pressure over both infra-orbital foramina; but the pain on the right side was evidently more severe and lasting than that on the left. The sites of both supra-orbital nerves were affected, and there was a painful and injected patch over the right malar bone, and another over the right lower canine fossa. The patient had all her faculties, except being rather deaf; arcus senilis was marked in both eyes, and the arteries were somewhat atheromatous. Urine 1015, acid, no albumen, no sugar. After an attack of pain there was free perspiration.

The operation was performed by me on February 25th, 1892, in the usual way as detailed above, and after the usual precautions. Chloroform was the anæsthetic employed, and I was assisted by my colleagues, Messrs. Barrow and Carless. The bleeding from the pterygoid veins was very free and copious, and considerable time was occupied in arresting it to a sufficient extent to continue the operation. Only one trephine opening was made at the spot represented in Fig. 12, and this was subsequently enlarged by the use of cutting-pliers and chisel and mallet. The edge of the trephine unfortunately wounded the dura on the outer segment of the circular incision, the skull being very thin at that spot. After the disc had been lifted out, and during the process of enlarging the opening, some cartilaginous tissue was removed, and on further investigation it was evident the Eustachian tube had been encroached upon. The dura was now separated from the skull around the opening, and the 2nd and 3rd divisions of the nerve traced up to the ganglion, and the posterior half removed. The wound was carefully irrigated, and the parts replaced in position with the exception of the coronoid process, which was snipped away.

The patient was considerably collapsed after the operation, the temperature falling to 97° F. At 4 A.M. on the following morning it rose to 101.2° , and she became restless and complained of occipital and frontal headache. The pulse became very rapid, varying from 120 to 130, the respirations increased to 40 per minute, and she gradually sank into a state of coma in which she died about forty-eight hours after the operation.

A complete *post-mortem* examination of the body was not permitted, but I opened the skull and was able to examine the base of it from within. The posterior half of the ganglion had been completely removed, the anterior half being surrounded by cloudy blood-stained serum, which on microscopic examination revealed the presence of pus corpuscles. There was considerable congestion of the right hemisphere, together with some softening of the temporo-sphenoidal lobe in the neighbourhood of the operation. I fear there can be no doubt that in this case the wound became septic in spite of all precautions, the opening of the Eustachian tube being possibly the cause of the infection.

I have recently (June, 1892) seen Case 4, and the woman, who is of a highly neurotic temperament, complains of a return of the pain in the temporal and maxillary regions. She flinches when these parts are touched; but on distracting her attention, the same parts can be handled with impunity. The tongue is still comparative anæsthetic on the right side. I do not consider that this case can be fairly looked upon as an instance of relapse, for there has been no return of the paroxysmal pain.

January 11th, 1892.

CASE OF CEREBRAL HÆMORRHAGE IN CALLOSO-MARGINAL FISSURE, WITH ANÆSTHESIA.

By THOMAS CHURTON, M.D. (Leeds).

MARY F., aged 66, was admitted into the Leeds Infirmary on September 18th, 1891, with right hemiplegia and partial anæsthesia, which had existed for three days. There was no aphasia. She was rather long in replying to questions; it seemed to be an

effort to comprehend them, but the replies were clear and normal. Her eyes were fairly bright; she was perfectly conscious, but easily tired by verbal examination. The urine contained one-twentieth albumen. A week after her admission, while investigating the degree and extent of the anæsthesia, I found that the dorsum of the *left* foot was completely insensitive, and that while a moderate pinch upon the right or paralysed side caused wincing and exclamation, and an apparently voluntary, but, perhaps, reflex, movement of the limb, upon the left side, where no paralysis had been noted or suspected, the sharpest pinch was not felt, and no movement followed. Higher up the limb the conditions were reversed, the pinch being felt on the left thigh and not on the right. Reflexes: plantar, slight on the right, very brisk on the left; patellar, sluggish on right, well marked on left. No ankle clonus. While examining the reflexes I did not perceive any drooping of the left foot. In the hands and cheeks sensation was more perfect on the left side. There could not be any doubt as to the results of the testing of the sensibility of the feet, but on attempting to go through the tests again she became dreamy and wandering, and it was impossible to keep her attention.

The history now obtained from her friends was as follows:—She was unmarried; had been a housekeeper until five years ago; but had since been living upon her savings, and dreading destitution. Of late she had seemed rather feeble, and had frequent “bilious” attacks. On September 15th, while visiting some neighbours, she suddenly complained of a numbness in the *left* leg, and began to rub the knee. Her friends were assisting her in this when, in about twenty minutes, she complained of the *right* leg also, and though still conscious, she was soon obviously partially paralysed on the right side. A few hours afterwards a slight difficulty of speech was observed, but there seems never to have been complete aphasia.

The observations upon the anæsthesia could not be repeated. On September 25th, the date of my examination, it was noted that the right thigh, though flaccid, was distinctly larger than the left, and that there had been pyrexia since admission—100—101°. This was due to right femoral phlebitis, which was followed by pulmonary phlebitis with hæmoptysis, and death occurred on October 2nd. No direct and precise observations, therefore, were made as to the power of voluntary movement in the left foot.

Undoubtedly she could, during her entire illness, move the left hip and knee; neither the house physician nor the nurses observed any weakness of the ankle; it did not droop, or strike any one as being paralysed; the clinical clerk (Mr. Mitchell), in fact, states from memory that the patient could move the toes of the left foot two days after admission.

At the necropsy there was found in the left hemisphere of the brain (1) a pool of subarachnoid fluid as large as a walnut, replacing the shrunken ascending parietal gyrus in its upper half. The ascending frontal gyrus seems to be of normal size. (2.) A flat sharply defined clot or disc of blood, 1 inch wide and $\frac{1}{8}$ inch thick, in the outer part of the optic thalamus and fibres outside it. There was a very small rupture into the lateral ventricle; and in the descending horn of the cavity was a solid darkish brick-red or maroon dry mass of blood-clot, the size of a large walnut. There was no fluid of any kind in the ventricle, and it was not a little surprising to find no erosion, no staining, softening, or obvious alteration of its surface, except at the bursting point above mentioned. On slicing the right hemisphere, a clot of the size of a small walnut was found in the calloso-marginal fissure. The convolution above the clot showed no erosion, discoloration, or signs of pressure. There was distinct discoloration of the convolution below the clot, *i.e.*, the gyrus fornicatus.

The hollow in which the clot lay measured about half an inch in depth horizontally. In this hemisphere there was no other lesion. The arteries at the base were thickly studded with milk-white atheromatous nodules like mustard seeds as to size. The spinal cord examined in its whole extent was normal. The right iliac vein contained a firm dark-red clot, which distended the vessel; the clot could be easily peeled off the lining membrane, but fine adhesive threads appeared to be broken in the separation. In the pulmonary artery, clots resembling that in the iliac vein were found in the later branches, chiefly on the left side, distending, but not distinctly adherent to, them. By an oversight, the pulmonary veins were not slit up. There was much blood in the lung parenchyma; microscopically, lacunæ, apparently caused by rupture of alveoli, were seen. Many of these lacunæ were even visible to the naked eye.

There seem to be in this case four points for remark: (1.) The double hæmorrhage. (2.) The femoral and pulmonary phlebitis.

(3.) The dry clot in the left lateral ventricle. (4.) The anæsthesia of the left foot in relation to the clot found on the median aspect of the right hemisphere.

(1.) In cases of apoplexy I have frequently observed several small hæmorrhages in other parts of the brain—meninges, white substance, pons—in addition to that which has been the fatal lesion. In a general disorder affecting the cerebral arteries more than one may arrive at the breaking point at or about a given moment. In the present case the excess pressure determining the rupture on the right side was probably caused by efforts at conversation; on the left, by the agitation and exertions following the first hæmorrhage. And (2) the occurrence of femoral phlebitis and afterwards of (probably) pulmonary phlebitis, shows that there was some cause of vascular inflammation at work within her. Judging by the number of cases of phlebitis in the wards, both medical and surgical, of the Leeds Infirmary, the disease has been unusually frequent during the past year. In one case there were the signs of pulmonary infarction (phlebitis or arteritis?) without any other vein being discoverably affected. And there is no apparent reason why inflammation should not originate in the pulmonary vessels, which are indeed much more prone to disease than they who are content with inspecting the first two or three inches only of the artery and vein would find reason to believe.

(3.) The dry, solid, brick-red clot in the left lateral ventricle, and the absence of erosion or other apparent change in the cavity, which after removal of the clot looked as if nothing remarkable, except some dilatation, had happened in it, can, perhaps, be explained by the supposition of a very minute rupture and a slight, but long-continued, trickling of blood. But the presence of phlebitis with similar clots in certain veins; the dry clot in a sulcus of the right brain; the curiously limited, flat, disc-like clot in or near the left optic thalamus suggest that there was present in the blood some toxine which caused a peculiar coagulation of the effused blood. Mr. Horsley was kind enough to send to me some years ago the heart of an animal into whose jugular vein he had injected iodine; in the right ventricle of that heart there was a clot (which had been formed instantaneously) very like the one found in this lateral ventricle. A few months ago I saw a very similar clot, about the size and shape of a fives ball lying loose in the left auricle of a patient who died from mitral valvulitis

(apparently *not* rheumatic), with secondary emboli in several arteries (femoral, cerebral, &c.).

(4.) The chief interest of the case, however, lies in the right hemisphere and the anæsthesia of the left foot. The want of positive observations upon the power of voluntary movement of the left ankle and toes at the time when the anæsthesia of the foot was discovered, detract, I fear, very much from the value of the case. But, though clots and tumours in sulci are not very uncommon, they often occur when attention is directed to other points, or in patients who cannot be rigidly investigated.

In one case a blood clot in the Rolandic fissure had caused brachial monoplegia during an attack of typhoid fever, the patient being a girl of 13; in the other, brachial monoplegia was caused by an enchondroma in the fissure secondary to growths in the knee and both lungs. In a third and more recent case, published by myself last year, tuberculosis of the paracentral lobule (old style) caused rhythmic twitchings, first of the thigh, and afterwards of the whole limb. As there was a large tubercular mass in the abdomen, formed by growth in prevertebral glands, it had at first been thought possible that some of the nerves in the vicinity had become involved, but the entire absence of any disorder of sensation was greatly opposed to this view. I used, it is true, no refinements in the examination of sensibility in this case, as at that time I did not know that it was seriously held that the Rolandic areas were sensory as well as motor. But in a case showing signs of motor irritation, the mere absence of pain or of any complaint of disordered sensation by a patient who was perfectly conscious, seems to negative the supposition that the area affected was sensitive in any marked degree. To one reflecting upon the facts already ascertained by Ferrier and others, it would seem most probable that the sensibility of the motor areas would be related to the motor function, to the instruments of motion; that the muscular sense, the sense of locality, and, perhaps, of pressure, would be felt in these areas, and, indeed, I find that Mr. Horsley has adopted—I do not know whether he originated—very nearly this view ('British Medical Journal,' November 3rd, 1888, p. 1008). But some maintain that tactile sensibility lies in the motor areas, and it has been proposed to excise the brachial motor area for obstinate recurrent neuralgia, beginning in the finger, and for which successive amputation, ending at the shoulder

joint, had already been practised. In such a case it is true that the motor area could be of no further use, and its excision could do no direct harm, but it seems unlikely that it would do any direct good.

The anæsthesia of the left foot in the case first related was not due to any local condition ; there was no œdema or other circulatory or trophic disorder in that limb. The evidence that it was due to injury to the right gyrus fornicatus beneath the clot, rather than to the motor gyrus above it, is—

(1.) No paralysis, such as must have accompanied the anæsthesia, if this had been caused by pressure upon the motor area, was observed.

(2.) The erosion or staining was upon the gyri beneath the clot, not on those above it.

(3.) The same motor area in another patient, irritated by tubercles in its substance, so as to cause somewhat violent movements, gave no symptom of sensory disorder.

Dr. Ferrier kindly permitted me to send the specimens I have described to the Neuro-Pathological Laboratory at King's College, and he has also favoured me with several criticisms and suggestions concerning the case, in addition to giving, with the assistance of Dr. Turner, some time to the complete examination of the brain.

Dr. ALDREN TURNER referred to the great interest which Dr. Churton's case had to the neurologist, both on account of the rarity of such cases and from the fact that it was corroborative evidence from disease in man of experiments made upon monkeys. He had only been able to find one similar record, in a case published by Dr. Savill in 'Brain,' Part 55, 1891. In this instance, left hemianæsthesia, associated with trophic skin changes, was due to an old hæmorrhagic lesion of the right gyrus fornicatus and neighbouring parts. Regarding the value of the case as a means of localising a cortical centre for common sensibility, he referred to the two prevailing theories on this point, firstly, that held by Munk and some other Continental investigators that the motor cortex of the Rolandic area was also sensory ; and, secondly, that originated by Dr. Ferrier, that the falciform lobe (gyrus fornicatus and gyrus hippocampi) represented a cortical sensory centre irrespective of any motor function. Dr. Churton's case, especially when taken in conjunction with that recorded by Dr. Savill, lent great support to the latter theory ; and it further seemed to indicate the existence of separate centres of common sensibility for individual portions of the body.

Dr. CHURTON, in reply, said that the frequent occurrence of multiple hæmorrhages in the brain was probably due to the existence of a generalised arteritis or phlebitis. This patient had two almost simultaneous cerebral hæmorrhages ; she also had femoral phlebitis and pul-

monary (phlebitis with) hæmorrhages ; it was probable that one virus caused all these inflammations. There was a moderate degree of atheroma in the arteries generally. The left posterior tibial artery had been specially examined : it was not occluded or even greatly diseased ; moreover, the presence of brisk plantar reflexes on the left side showed that the nerves were well nourished, and that the anæsthesia was not due to loss of blood-supply. There were no trophic changes in the left leg : it was not even observed to be cold. The spinal cord, examined throughout, was normal.

THE RADICAL CURE OF PROSTATIC OBSTRUCTION BY THE GALVANO-CAUTERY.

By W. BRUCE CLARKE, F.R.C.S.

OF the various surgical procedures which have during the last few years been proposed for the relief of prostatic enlargement, most have been directed towards the removal of the gland itself, or some portion of it, either through a perineal wound or after the bladder has been opened by the suprapubic route. When a considerable amount of enlargement exists so that a swelling the size of a small egg can possibly be felt *per rectum*, either with or without the introduction of an instrument into the bladder, such operations as these are not only useful, but are urgently called for. But the conditions which demand them are comparatively rare. In by far the great majority of cases, the obstruction to urinary outflow is produced by some small portion of the prostate gland overlaid, perhaps, by a piece of swollen and inflamed mucous membrane. Indeed the obstacle may be so slight that it is oftentimes overlooked after death, unless it is very carefully sought for at the *post-mortem*.

Even in those cases in which the prostatic obstruction is accompanied by a considerable amount of prostatic hypertrophy, it is very often the case that the actual cause of obstruction is some tiny portion of the gland, and the large mass, which is so obvious when the bladder is opened, has played but a small part in obstructing the urinary outflow. It is clear then that no operation, which does not aim at making more patent the actual orifice of the prostatic urethra, is likely to succeed in restoring the patient to natural urination. Some few years back, Mercier, of Paris, attempted the removal of the offending portion of the gland by means of an instrument shaped somewhat like a lithotrite, by

which means he punched out the obstructing portion of the gland and mucous membrane. In many instances the results which he obtained were excellent for a few weeks after the operation was performed, but relapse speedily took place, and no permanent benefit ensued. I have myself tried this instrument on several occasions, but success in each instance was but temporary, and in one of them by no means complete even for a time. A short time back the same problem was attacked by Professor Bottini, who has made use of the galvano-cautery, and, after numerous experiments both upon living animals and upon dead tissues, he has succeeded in constructing an instrument, his "*cauterisatore termogalvanico*," which is here displayed. In shape it resembles an ordinary lithotrite, or, to be more precise, the instrument of Mercier, and consists of a metal tube which is divided into two compartments by means of a thin septum. By this contrivance a continuous stream of water can be passed through, and thus ensure that it will not become too hot during an operation or inflict any damage on the rest of the urethra.

The galvanic current is conducted to the cautery by means of a wire which is thoroughly insulated, and passes down the inside of the tube, whilst the metal of the instrument is utilised for the return current. The cautery itself consists of a slip of thin platinum about $\frac{3}{4}$ inch in length and $\frac{1}{8}$ to $\frac{1}{4}$ inch in breadth, which is laid upon a piece of porcelain. This is sunk in the bend of the instrument so that it does not stand up from its surface, or in any way impede its introduction into the bladder, or its withdrawal from it. It is so placed that when the beak of the instrument is turned round towards the rectum the platinum plate rests on the obstructing portion of the prostate gland. Before introducing the instrument it is, of course, essential to ascertain that a sufficient current is available to heat the platinum to a dull red heat, by which means the tissues can be destroyed, without the occurrence of any serious bleeding. The period during which the heated instrument is kept in contact with the prostate is, as a rule, only fifty seconds, after which it is carefully withdrawn, and a soft red rubber catheter is introduced into the bladder and retained there for forty-eight hours. Great care is necessary both in the removal of the one instrument and in the introduction of the other, so as not to displace the eschar which has been produced by the cautery. Bottini himself does not in all cases

employ chloroform, as he finds that the operation is by no means a painful one. In my operations, however, the patient has always been anæsthetised, partly in order to avoid pain and partly for the sake of ensuring that perfect quiet which is so essential to a successful result. So much depends on an accurate adjustment of the instrument and on bringing the cautery into exact apposition with the obstructing portion of the prostate, that every means should be employed of putting the patient into a position which will enable the surgeon to gauge as precisely as possible by his sense of touch the exact locality of the obstruction. And it is no less important that when the instrument is once in action, no dislodgement should occur, which quite possibly might be the case were the patient not somewhat deeply anæsthetised.

As a rule but little, if any, pain is experienced by the patient when he comes round from the anæsthetic. Often until the catheter is withdrawn forty-eight hours later but little pain is complained of. It is usually not until the instrument has been removed for a day or two that the eschar begins to separate, and its fragments are brought away with the urine, generally without any difficulty. This process continues usually for about a week, and during this period there is some liability to blocking of the urethra by the sloughs, which necessitates the careful passage of a catheter. A week or two later when the urine begins to get clear and free from slough, it is advisable to gently introduce a catheter for the purpose of ascertaining whether any residual urine still exists in the bladder. The rapidity with which the bladder will regain its power of expelling urine must, of course, depend upon the extent to which atrophy of the muscular wall has already taken place, and to a certain extent upon the presence or absence of previous cystitis. But it is remarkable how soon, provided the patient is fairly healthy, complete recovery takes place. It not unfrequently happens that for a few days after the operation the patient is unable to hold his water satisfactorily, but this is only a temporary condition and speedily passes off. In one of my cases the patient had suffered with urinary trouble, frequency of micturition, &c., for about a year, and had 18 ounces of residual urine when he first came into the hospital, and yet in less than three weeks he was walking about, able completely to empty his bladder, and has remained perfectly well up to the present time, now more than six months since the operation. The follow-

ing notes will serve to indicate the course and progress of this plan of treatment :—

CASE 1.—W. P——, aged 73, came under my care in St. Bartholomew's Hospital on June 20, 1891, complaining of urinary incontinence. He stated that for the past year or more he had noticed that his water had not passed so freely as it should, but he had always been able to void it until April of the present year, when after a day's work, which involved some prolonged stooping, he was quite unable to make water, and had to send for a surgeon to relieve him. A catheter was passed without any very considerable trouble, and he believed himself to be all right ; but the next day his testicles began to swell, and he was obliged to go to bed. He suffered a good deal of pain in the testicles for several days. During the last two months he has been unable to retain his water, which has dribbled away from him, particularly at night. He has not slept well recently, and has become a good deal thinner.

There is at present a slight enlargement of the prostate to be felt *per rectum*, and a metal instrument does not enter the bladder without some difficulty.

Between his admission and the 8th of July, his residual urine was drawn off regularly twice a day. It varied between 4 and 7 ounces in amount.

July 8th. Under chloroform ; Bottini's instrument was introduced into the bladder, and connected up with two accumulators, which it had been previously ascertained would heat it up sufficiently. The instrument was turned round so as to bring the platinum into relation with the obstructing prostate, and the current was turned on for a period of 50 seconds, after which it was withdrawn, and a No. 10 red rubber catheter introduced and tied in. Dr. Marotti (Bottini's assistant) was present both at this case as well as at the succeeding one, and I feel that I am much indebted to him for the care with which he explained and demonstrated the various points of the method, and without his assistance I am sure I should not have been able to succeed as I have done.

July 9th. The next morning the catheter slipped out, but the house surgeon, Mr. Maund, found no difficulty in introducing a coudé in its place.

July 10th. Patient expresses himself as feeling quite comfortable. Urine acid ; contains some amount of phosphates and pus. The catheter slipped out a second time, but was easily replaced.

July 13th. Catheter withdrawn, as it seemed to be causing a good deal of irritation, and the temperature had risen to 100° F. Feels comfortable, and passes his water without much inconvenience.

July 27th. During the last fortnight he has had no special trouble, but his right testicle has swollen a good deal, and to-day there is evidently some pus in connection with the epididymis and tunica vaginalis. Pus let out under gas and ether. Boracic fomentation.

August 18th. Patient's general condition good. He eats and sleeps well. Does not get up at all at night to pass water, and can hold his water during the day for four or five hours continuously. A No. 12 catheter was passed, and no residual urine whatever was found in his bladder. The wound in his scrotum is well.

December 28th, 1891. Letter from patient to say he is perfectly well, and able to do his work. A catheter was passed in October for diagnostic

purposes, and there was then no residual urine whatever. Well, June 10th, 1892.

CASE 2.—J. P——, aged 58, admitted into St. Bartholomew's Hospital on August 17th, 1891, suffering from difficulty in micturition. He is a healthy-looking man, and states that he has had trouble with his water to a marked extent for the last three months, and of a slighter nature for eight or nine months more. At the present time he has to get up two or three times a night to pass his water, and only can get rid of it after a great deal of straining.

On first coming into the hospital, a No. 12 English instrument was passed, and, after he had emptied his bladder as far as he could by himself, 14 ounces of residual urine were drawn off. During the next few days the residual urine varied from 9 up to 17 ounces: the water was perfectly clear, acid, and containing no albumen; sp. gr. 1018.

August 24th, 1891. Operation: after the patient had been placed under the influence of an anæsthetic, and the bladder had been completely emptied of urine by means of a catheter, Bottini's instrument was introduced into the bladder, and the beak was reversed and pulled back until it was fixed against the obstructing prostate, the instrument being placed at such an angle as to ensure this. Connection with the battery was then made, and continued for fifty seconds, cold water circulating through the hollow portion of the instrument at the same time. The instrument was carefully withdrawn, and a No. 14 soft, red rubber catheter introduced on a stilette. The stilette was withdrawn, and the catheter left behind. The patient complained of but little pain during the next day or two.

Forty-eight hours later the catheter was withdrawn, and he passed his water perfectly well.

August 28th. He has to strain a little to pass his water. During the night he passed a few sloughs. Urine rather thick, acid; sp. gr. 1020.

August 29th. Passes water quite easily without straining.

September 3rd. A No. 11 red rubber catheter was passed. A little obstruction was found near the prostate, but only 1 ounce of residual urine could be drawn off.

September 7th. Still passing one or two tiny sloughs, which cause occasional straining, otherwise quite well; no residual urine. Urine contains some phosphates, acid; no albumen or sugar.

Seen on two occasions since, and a catheter passed. No residual urine. Feels perfectly well.

Letter, December 29th, saying he is still quite well.

June 4th, 1892. Still quite well.

CASE 3.—H. G. S——, aged 67. (Sent to me by Dr. Harper, of Kensington.)

July 2nd, 1891. First came under my care suffering with a stricture, and complaining that he could not get rid of the whole of his urine without the aid of a catheter. He had had his stricture divided some few years previously, but partial recontraction had taken place, and only a No. 6 would pass through it. There was only a slight amount of prostatic enlargement to be felt *per rectum*, and no difficulty was experienced in passing the catheter into the bladder. Under these circumstances treatment of the stricture was advised in the first instance, it being explained to the patient that his prostate might require treatment later. The stricture was treated in the usual fashion by electrolysis, and, notwithstanding

several slight attacks of gout which induced a great deal of vesical irritability, by October 7th a No. 16 English easily passed into the bladder.

The water was clearer than it had been, but the residual urine still continued, and varied in amount from 4 to about 8 ounces.

November 30th, 1891. Dr. Harper being present, and Mr. Mills administering the anæsthetic, whilst Dr. Lewis Jones assisted with the accumulators, Bottini's operation was performed exactly in the same way as in the last two cases, and a red rubber catheter tied in.

December 1st. The temperature rose to 100·4, and there was a good deal of vesical irritability.

December 2nd. The instrument was withdrawn and a good deal of mucus came away, with perhaps a dessertspoonful of purulent-looking fluid. The urine was only slightly acid, as large doses of citrate of potash had been administered in order to diminish the acidity, which seemed to aggravate largely the vesical irritability.

December 5th. The patient was able to pass his water in the standing position, which he had been unable to do for more than a year, but, though much clearer than it had been, the water was still thick, and contained a good deal of ropy mucus. He had been for several hours the day before in a good deal of pain, owing to the slough sticking in his urethra, and it had only been dislodged by the passage of a catheter.

December 21st. Has gone on uninterruptedly well since the last visit. Sits up and goes about the house as usual. He has so long been accustomed to the use of his catheter that he is perhaps inclined to use it too often, and provoke a certain amount of extra vesical irritability.

January 11th, 1892. He seems in excellent health: goes out in all weathers. There is still some residual urine, but slightly less than there was. Occasionally some ropy mucus in the water.

Passes water with perfect ease in the standing position.

CASE 4—J. C——, aged 72. Admitted under my care in St. Bartholomew's Hospital on November 14th, 1891, suffering from retention of urine.

He has had difficulty with his water for the last fifteen or twenty years, and has from time to time been obliged to seek medical aid and have catheters passed. His water is only passed after considerable straining, which has been at times so severe as to cause some prolapsus ani. At the present time he suffers quite as much, if not more, with his rectum than with his bladder. For the last week he has had to have his water drawn off with a catheter twice daily, and there has been at times a good deal of blood mixed with it.

On admission. A great deal of pain. 16 ounces of water withdrawn by catheter. Contains some blood. Morphia injection, $\frac{1}{4}$ grain.

November 18th. Can only pass a very little water: most of it has to be drawn off. A good deal of pain still. The urethra is exceedingly sore. Temperature 104·6°. Rigor. Urine acid, 1018. No albumen. Contains some blood.

November 26th. He is now much better, passes a good deal of his water unaided, but there is about 8 ounces of residual urine.

December 8th. Under an anæsthetic, the residual urine, amounting to about 6 ounces, was drawn off. Bottini's galvano-cautery was passed into his bladder, and the current turned on for the space of 50 seconds, after which the instrument was withdrawn and a No. 12 red rubber catheter was introduced. About half an hour after the operation the patient passed about 3 ounces of very blood-stained urine, and complained of a

good deal of pain. During the night he suffered from a desire to micturate about every half hour.

December 9th. In his attempts to micturate he managed to pull his catheter out, which was not replaced, and he has been much more comfortable ever since.

December 10th. Slept much better and feels easier ; says he has not passed water so easily for years.

December 11th. A large slough, measuring nearly three-quarters of an inch in length, has passed with his urine, which, with the exception of the sloughs, is quite clear, acid, 1020 ; no albumen.

December 17th. Catheter passed after he had micturated. There was no residual urine whatever.

December 18th. His prolapsed rectum and piles were operated on, a cautery being used to the mucous membrane.

December 19th. Had to have his water drawn off.

January 7th. He is now perfectly well, passes his water without any difficulty, and has no residual urine. Piles and prolapsus are also well.

From the notes which have just been read it will be seen that the operation in question is one which has, so far, yielded most excellent results, in a class of case which is usually amongst the most intractable in surgery, so far as cure is concerned. The only one of the four cases in which the temperature after the operation rose as high as $100\cdot5^{\circ}$, or gave one the slightest momentary anxiety, was the first, in which case also it will be remembered that there was an abscess formed in connection with the testicle, thus unduly prolonging the period of convalescence. To a large extent, if not entirely, this is, in my opinion, due to the retention of the catheter for a longer period than forty-eight hours.

It is the practice of Professor Bottini to insist on this plan of procedure ; but such retention is, I am convinced, both unnecessary and undesirable, as is shown by the more favourable course which the other cases have taken.

As far as the results are concerned, three out of the four cases now pass the whole of their urine without catheter, and experience no difficulty whatever in its passage, whilst the fourth, which was the most severe case of them all, is so far improved that he can urinate when standing up, which he had been unable to accomplish for some year and more, as well as having much less residual urine in his bladder than was formerly the case.

Mr. HURRY FENWICK stated that he was unable to view the Bottini operation in the same roseate light which Mr. Clarke evidently regarded it. He had employed the method both at the London Hospital and at St. Peter's, having obtained the instrument and instruction in its use from Professor Bottini's assistant, Dr. Marotti, in the early part of last

year. It was too soon, however, to mention any apparent success which he had obtained with it, and he thought that the discussion ought to run on the lines of: 1. Is the instrument a practical one? 2. How far is the method applicable to obstructive prostatic enlargement? For his part, he considered the instrument far from perfect. His own instrument had to be sent back to Pavia on account of the deficient insulation. Moreover, it was long before sufficient electromotive force could be obtained to make the platinum plates hot enough. Krohne and Sesemann had sent especially to Italy for Professor Bottini's battery, and on its arrival it was found to be useless. Mr. Fenwick had instructed Leiter, of Vienna, to make him an instrument on somewhat the same lines, and this new pattern he had used with greater confidence than the original one. It differed in every detail from the Bottini type, though the same in principle, and he was told that it required half the current force. (Both patterns were shown.) 2. How far was the method of use? The method was obviously of no use in cases of enlarged median lobe in the form of a tumour of any size, and this fact was impressed upon him lately by finding himself forced to remove a small chestnut-sized growth of the middle lobe through a suprapubic opening, after he had attacked it with the Bottini method two weeks before, but without result. Now it had been shown by Watson, of Boston, that in twenty-seven of the thirty cases examined by him after death, a median enlargement existed. It can be therefore asserted that the Bottini operation is inapplicable in over one-third the cases. This proportion in actual practice is about right. After a very careful unbiassed trial of the method, Mr. Fenwick was of an opinion that the operation would only be of use in those cases in which a bar of prostatic tissue existed at the neck of the bladder and formed the chief obstruction to the outflow of urine. He believed the instrument would be used by few surgeons, on account of the cumbersome impedimenta necessary to work it, and most surgeons, he thought, would object to place so formidable and so dangerous a cautery on the prostatic neck without either perineal drainage or perineal control of a possible hæmorrhage.

Mr. SWINFORD EDWARDS congratulated Mr. Bruce Clarke on the success this method had met with in his hands. He considered that the cases had been selected with wisdom, on account of the freedom from cystitis and even worse complications, after the application of the cautery. He had not tried this method himself, though in several cases he had met with temporary success after punching out a portion of the obstructing lobe or bar by means of Gouley's prostatome. As obstruction to micturition recurred generally in a few weeks in his cases, he was glad to hear that the improved condition of Mr. B. Clarke's patients was of longer duration. It was, as yet, early days to talk of a permanent cure. Mr. Edwards drew attention to the electrolytic puncture of the prostate through the rectum, as advocated by Dr. Casper, of Berlin, but at present did not feel inclined to recommend it. He said that cases of enlarged prostate requiring treatment other than by catheterism were, as a rule, bad subjects for operation, being broken down in health and the possessors of damaged kidneys. Therefore, the less severe the operation, the better chance had they of pulling through. If by this method normal micturition could be re-established, a lasting boon would be conferred on both patients and surgeons.

Mr. BUCKSTON BROWNE said that the ordinary prostatic case was usually such an exceedingly simple one to manage that we should be very

careful before we rushed to new methods of treatment. He regarded suprapubic prostatectomy as dangerous, and only to be resorted to in extreme cases. He pointed out, however, that there were indications for its employment. The operation of Bottini was enticing from its apparent simplicity, and was therefore apt to be employed in unsuitable cases. A man who had only just begun to use a catheter should certainly not be submitted to such an operation. The fourth case appeared to him to be one of bad stricture together with atony of the bladder; he thought it probable that after treatment of the stricture the bladder would have recovered power independently of Bottini's operation, and the good result therefore ought not to be credited to the latter. It should not be forgotten that it was quite possible for a man to regain power absolutely over his bladder after having lost it for a considerable time. He related an instance in which a gentleman, being unable to relieve his bladder, was left afterwards in a condition of retention for sixty hours. Three pints of urine were then drawn off, but for six months afterwards he could pass no urine except by catheter. He was now, however, able to empty the bladder entirely by natural effort. He agreed with Mr. Fenwick that the majority of cases of prostatic obstruction were due to middle lobe enlargement, for which this procedure would be of no avail; in such cases, incision from the perineum or urethra met with but trifling success.

Mr. CLARKE, in reply, said he had carefully selected his cases. He had already stated that, in cases of middle lobe or of irregular enlargement, if any treatment beyond catheterism were indicated, then the suprapubic operation should be done; he had not, of course, advocated Bottini's method for these. One could not expect to get a good result unless the bladder were healthy, and he doubted if the operation were more dangerous than lithotomy in skilful hands. He admitted that an instrument requiring a lighter battery power could be constructed, and would be more convenient; but that did not touch the question of applicability of the method. He held that the question of frequency should not be decided by an appeal to museum specimens; the only statistics of value were those of the *post-mortem* room.

February 8th, 1892.

ENLARGEMENT OF THE SPLEEN IN YOUNG CHILDREN.

By J. WALTER CARR, M.D.

As in adults, so in children, enlargement of the spleen may be due to numerous and well recognised causes, some of an acute, others of a chronic, character. Typhoid fever, malaria, leucocythæmia, tuberculosis, embolism, lardaceous disease, cirrhosis of

the liver—each one of these may of course produce a greater or less hypertrophy of the spleen. But whilst in very young children enlargements due to any of the above-mentioned conditions are comparatively rare, and not usually difficult of diagnosis when they do occur, there is a splenic hypertrophy found in infants, very chronic in its course, very serious in its prognosis, so frequent in its occurrence as probably to out-number all the cases of enlarged spleen due to other causes during infancy, and yet so ill-defined in its etiology that it has hardly acquired a distinctive name, and in many medical works is only referred to incidentally under the heading of rickets or of syphilis. Nevertheless, its course, symptoms, and pathological anatomy appear to be so definite and characteristic as to constitute it a disease *sui generis*, and entitle it to as well-defined a place in our classification as has been assigned to the allied conditions leucocythæmia and lymphadenoma, from which, however, as we shall presently see, it is easily distinguished. Perhaps the term “splenic anæmia,” proposed by Griesinger, is the best to assign to it. It is necessary to distinguish a spurious from a real enlargement of the spleen in children; sometimes, without any genuine hypertrophy, it is considerably longer and thinner than natural; under such circumstances, particularly should it be pushed down by the contraction of a rachitic thorax, the lower end may be felt below the ribs, but it feels soft, not hard; there are none of the characteristic symptoms associated with genuine hypertrophy, and several times at a necropsy I have found that, though considerably elongated, the weight of such a spleen has not been abnormal or its consistence increased.

The following description of the symptoms and course of the disease is based on thirty cases observed during the last three years at the Victoria Hospital for Children, Chelsea. During that period not a single example of genuine leucocythæmia came under my observation, and the cases of enlarged spleen from all other causes (chiefly tubercular) in children under 3 years of age were fewer in number than those of simple splenic anæmia. Sixteen of the patients were males and fourteen females; in age they varied from 2 months to $2\frac{1}{2}$ years when first seen. They were usually wasted, but by no means invariably so, particularly the slighter and earlier cases; but only in one or two instances were the children really plump and well developed. There was nearly always some pallor; this was extremely marked in the more severe

cases, and was associated often with the more especially characteristic splenic appearance—a waxy colour with a faint olive tint, indicating almost at a glance the nature of the case. The degree of enlargement of the spleen varied; in nearly every instance it could be distinctly felt at least an inch or an inch and a half below the costal margin, and in about half the cases it extended down to the level of the anterior superior iliac spine and forwards nearly to the umbilicus. It always felt exceedingly hard, and was not usually tender. It must be remembered that in children the costocolic fold of peritoneum is much firmer than in adults; so that in the earlier stages of splenic enlargement it directs the lower end of the organ forwards, and so prevents it from projecting as far below the ribs as it otherwise would, and perhaps causes it to encroach more on the thoracic cavity. As a rule, the larger the spleen the more marked was the anæmia, but to this there were some marked exceptions; thus in Cases 20 and 24 the spleen was large but the anæmia slight, and *vice versâ* in Cases 4 and 10. The liver was apparently enlarged in about half the cases; but little importance can be attached to the extent of its projection below the costal margin, on account of the varying contraction of the chest by rickets, and the weight of the organ *post mortem* never indicated any decided hypertrophy. There was usually also swelling of some of the external lymphatic glands, but never to any marked extent; they were always hard and freely movable. In four of the fatal cases (8, 15, 16, and 30) hæmorrhages occurred—either epistaxis or small scattered purpuric spots in the skin, or both—and Case 6, the child recently under treatment in Westminster Hospital, had profuse epistaxis shortly before admission there. These hæmorrhages probably depend solely on the severe anæmia, for, as Dr. Stephen Mackenzie pointed out in his Lettsomian Lectures on Anæmia in 1891, they may be met with whenever the proportion of red blood-corpuscles falls below 50 per cent. of the normal, a condition which obtained in all the cases referred to. In a similar way may be explained irregular attacks of moderate pyrexia, which occurred from time to time in the more severe cases whilst under observation in the wards. There was seldom an opportunity of examining the urine, but in two patients (Cases 15 and 30) a faint cloud of albumen was present, and in three others (Cases 5, 6, and 7) it was noted to be absent. According to Dr. Crozer Griffith, in Keating's 'Encyclopædia of Diseases of

Children' (vol. iii, p. 799), the urine in this disease does not contain albumen.

The blood was examined, once or oftener, in fifteen of the children, and showed merely a condition of simple anæmia. In the most severe cases it was distinctly watery and paler than natural, and flowed very freely from a small prick. The red corpuscles always ran into rouleaux; they varied considerably in size, but presented no abnormal shape. In number they ranged from 32 to 78 per cent. of the normal; in only three instances, however, was the percentage less than 40, and two of these were fatal; the general range was from 45 to 70 per cent.; and even this may give somewhat too low an average, as in several cases in which the blood was not examined the anæmia was not at all profound. In one infant (Case 8) the number of red discs fell in eleven weeks from 64 per cent. to 37 per cent., then under treatment with reduced iron rose again to 63 per cent., when the patient died of diphtheria. In another instance (Case 20) which steadily improved, the percentage rose from 62 to 82·6. The percentage of hæmoglobin was ascertained in seven of the cases, and was not found to bear any definite relationship to the number of red corpuscles; in all, however, as in simple anæmia and chlorosis, and in contrast with pernicious anæmia, it was deficient, both actually and also relatively to the number of corpuscles. The hæmoglobin value of the individual corpuscle was usually from 50 to 67 per cent., but in one case (19) in which the percentage of red discs was 78, two estimations of the hæmoglobin gave only 25 and 28 per cent. respectively. From the few cases in which several estimations were made the increase or decrease of hæmoglobin seemed roughly to vary *pari passu* with that of the red corpuscles. It is perhaps doubtful whether the results of examination of the blood with the hæmoglobinometer are very reliable in childhood, and I shall be glad to hear the opinions of others on this point. In nearly every instance there was a slight excess of white corpuscles, but this was not marked, especially when we remember that in young children they are normally somewhat more numerous relatively than in adults—in infants, 1 white to from 130 to 210 red corpuscles. In only one instance (Case 8) did the percentage of white discs approach the leucocythæmic condition, and that was after epistaxis, when the red corpuscles fell to 37 per cent., with 1 white to 33 red ones. In this case all the lymphatic glands,

both internal and external, were enlarged, though none were bigger than a small filbert; even the Peyer's patches and solitary glands of the intestine were found distinctly swollen. In the other cases there was 1 white to from 70 to 225 red discs, averaging about 1 to 100, so that it is quite evident that this disease is distinct from leucocythæmia.

It is well known that anæmia is often a prominent symptom of rickets, and, as nearly all these children were rickety, the question naturally arises whether the blood changes described were dependent mainly, or even entirely, upon the rickets. It seems to me, however, that, at any rate in the marked cases, the anæmia was always more profound than could be explained by the rickets alone, and, more particularly, that the diminution of red corpuscles does not occur to such an extent in simple rickets. Thus, in one case of well-marked rickets and anæmia, I found 98 per cent. of red discs and only 28 per cent. of hæmoglobin, and in twins with marked rickets and pallor, the percentage of red corpuscles in each was about 80 and of hæmoglobin, 70. As I have already stated, in none of my cases of splenic enlargement did the percentage of red discs exceed 78. Moreover, in the blood of cases of simple rickets there does not seem to be the same tendency to even a slight excess of white corpuscles.

The course of the disease is essentially chronic, and so it is difficult in out-patient practice to trace the cases to their termination. In some instances the splenic enlargement and the anæmia steadily increase, hæmorrhages occur, and the child dies at last from exhaustion, usually complicated by more or less catarrh of the mucous membranes, leading to bronchitis and broncho-pneumonia, or to diarrhœa. Ten of my thirty cases are known to have died mainly in the way just mentioned; but this is, perhaps, a higher mortality than that of a number of cases taken indiscriminately, as the thirty cases include an undue proportion treated as in-patients, on account of their specially severe symptoms. Six of the cases could not be traced; of the remainder, thirteen were found either to have completely recovered or to be steadily improving; Case 6 alone remained *in statu quo*. In Case 3, after only four months of treatment, the child's general condition and colour were greatly improved, and the spleen could scarcely be felt, but this was a slight case from the first, and the spleen was never much enlarged. In Case 10, examined after a year and eight

months, the spleen could not be felt, and the red discs had risen from 42 per cent. to 104 per cent. In Case 19, after eight months, although the spleen was still enlarged, the red corpuscles had risen from 78 per cent. to 98 per cent., the hæmoglobin from 26 per cent. to 45 per cent., and the white corpuscles, which had been in the proportion of 1 to 100 red, were now only 1 to 245. In Case 20 the spleen at first reached to the level of the anterior superior iliac spine, and the anterior border was in the nipple line; the lower border of the liver was felt half-way between the umbilicus and the ribs; the inguinal glands were enlarged. Five months later the child was stouter and of better colour, the spleen could not be felt, the liver only projected an inch below the costal margin, and there was no glandular enlargement. From the first, however, the anæmia in this case was not extreme, viz., red discs, 62 per cent., hæmoglobin, 45 per cent.; the former rose to 82·5 per cent., the latter remaining about the same. In Case 22 the spleen was nearly as large as in the one just described; after three months it only reached an inch below the ribs, and there was much improvement in the child's colour and nutrition; it unfortunately succumbed to an attack of whooping-cough. Occasionally there seems to be no definite change after several months of treatment; Case 6 shows this especially, as after two years the child's condition was almost unaltered, the anæmia much the same, and the spleen a little larger.

As is usually the case in anæmia when improvement occurs, the percentage of red corpuscles rises to the normal before that of hæmoglobin.

The prognosis of cases of splenic anæmia as given by different writers varies immensely. Crozer Griffiths, in Keating's 'Encyclopædia,' says that permanent cures are of the rarest occurrence unless the splenic enlargement be due to syphilis or malaria; Henoch says that most of the children die from progressing anæmia, wasting, and, finally, dropsy of the cavities; Eustace Smith, on the other hand, gives a much more favourable prognosis; whilst Ashby and Wright state that most of the cases improve after a while, but that their course is very chronic. From my own observations I should say that the prognosis in these cases is not necessarily bad, though the disease will probably be of long duration; but should the spleen get very large and the anæmia profound, there is, of course, great danger of death from

simple asthenia, from pulmonary trouble, or from the onset of any acute specific disease, which will probably prove rapidly fatal in a child already greatly weakened. Although the prognosis is usually more unfavourable, the greater the enlargement of the spleen, yet it is interesting to note that in Case 4, in which the child steadily got worse and had marked anæmia, the organ was only slightly enlarged, weighing 1 ounce. Later on I shall mention facts which seem to point to the presence of well marked syphilitic manifestations being a particularly unfavourable indication.

Morbid Anatomy.—A necropsy was obtained in 7 (Cases 4, 8, 15, 16, 26, 27, and 30) of the 10 fatal cases. In all there were great emaciation and pallor of the internal organs, in 4 marked bronchitis or broncho-pneumonia. Case 4 was the only one in which there was any sign of tubercle; in this case there was a mass of caseous mesenteric glands. The liver was but slightly, if at all, enlarged in any instance, its greatest weight being $15\frac{1}{2}$ ounces. In appearance it was either normal or paler than natural; in only 2 of the cases (4 and 26) was it notably tougher than usual, and in 2 others (8 and 15) the consistence was very slightly increased. In 1 case (8) there was a depressed scar-like patch in the liver, perhaps the remains of a gumma; this was the only indication of visceral syphilis found in any examination. We may conclude, then, that the changes which the liver undergoes in these cases are unimportant, and that its enlargement is apparent rather than real, being due either to the relatively larger size of the liver in infancy or to the contraction of a rachitic thorax.

As regards the internal lymphatic glands, in no case was there any swelling of those in the thorax beyond what would be attributable to bronchitis or broncho-pneumonia. In 4 cases the mesenteric glands were normal; in 1 case, as already mentioned, caseous; and in 2 (Cases 8 and 16) slightly swollen. The bone marrow I have not examined, but it is said that that of the long bones may present in some cases the same dark-red lymphoid appearance frequently seen in pernicious anæmia.* In 2 cases the spleen was slightly adherent to the surrounding parts, and in a third there were a few flakes of lymph on the surface, but in no

* Dr. Crozer Griffith's article on "Splenic Anæmia," in Keating's 'Encyclopædia of Diseases of Children.'

instance was there any distinct thickening of the capsule. In 1 case (4) the organ was but slightly enlarged, weighing 1 ounce; in the others it varied from 4 ounces to $8\frac{1}{2}$ ounces in weight. In all it was firm, and the consistence was distinctly increased; the section was dark and fleshy, but otherwise fairly natural in appearance, with no enlargement of the Malpighian corpuscles. Iodine solution gave no albuminoid reaction. None of the other organs presented any noteworthy change. The microscope confirmed the naked eye appearance of the spleen by showing that the enlargement was apparently due to a simple hypertrophy, with more or less increase of fibrous tissue. In 1 case (8) there were scattered patches having somewhat the aspect of albuminoid change, a condition probably identical with the hyaline fibrosis which Cheadle has described.* The microscope showed no important changes in the liver or lymphatic glands, but in some instances there seemed to be slight overgrowth of connective tissue in the kidneys. The appearances presented by the spleen seemed to be the same whether the enlargement were associated with syphilis, with rickets, or with neither of these diseases; it also resembles the ague spleen. The condition described corresponds with the descriptions given by other writers, especially by Henoch and by Ashby and Wright; but I cannot be sure whether Jenner refers to a similar condition, when in his lectures on rickets he speaks of an albuminoid infiltration of the spleen, with some similar affection of the liver and lymphatic glands, leading to enlargement of all these organs, and causing the emaciation seen in some cases of rickets. He describes the spleen as non-adherent, capsule scarcely, if at all, thickened, the substance of the organ tough but elastic, pale-red or dark-purple in colour; liver usually only slightly enlarged, heavy, tough, and semi-transparent; external lymphatic glands enlarged, not tender, and never inflamed. Although he speaks of an albuminoid infiltration of all these organs, he says that it has not the characters of the albuminoid change described by Virchow, and especially that it does not give any reaction with iodine solution. On the whole, from the general symptoms and accompanying conditions of these cases, it seems probable that the so-called albuminoid infiltration of the spleen associated by Jenner with certain cases of rickets is identical with the condition which I have been describing, but represents probably a more

* 'Brit. Med. Jour.,' vol. ii, 1888, p. 1148.

advanced stage of the disease, and one which does not seem to be commonly met with.

I come now to the most important part of the subject—viz., the pathology of this enlargement of the spleen. Three diseases naturally suggest themselves as possible causes—ague, congenital syphilis, and rickets. We will discuss them in the above order. Ague is now so comparatively rare as an indigenous disease in London that one is apt to overlook it as a still possible cause of obscure conditions, and it is very necessary to bear in mind that although we may no longer meet with well-defined attacks of intermittent fever, yet malaria is much more insidious in its invasion in children than in adults, and may conceivably be the basis of the condition which has been described, especially as most of the patients came from the low-lying districts of Chelsea, Battersea, Fulham, and Putney, adjacent to the Thames. In vol. i of 'The Lancet' of 1869, p. 325, is a statement by Dr. Andrew, of St. Bartholomew's Hospital, that almost every autumn he got some cases of ague which had originated in certain streets in Blackfriars. At a discussion at the Medical Society on leucocythæmia, as lately as 1880,* Sir William Gull stated that there was malaria all over London; and in 1884 appeared a paper by Dr. W. R. Thomas, of Sheffield,† describing cases of malaria, curable by quinine, amongst children in the low-lying districts of Sheffield, in which the spleen was generally more or less enlarged, sometimes enormously so. Moreover, malarial fever may be hereditary, at any rate from the mother. Henoch‡ mentions a case of splenic anæmia in which the mother stated that she had repeatedly suffered from intermittent fever during her pregnancy with the patient. In my cases the only hereditary histories obtainable were in Case 12, the father having had slight ague in Essex when a boy, and Case 15, in which the father had had ague twice, when in the army, before marriage; but probably during that period he had also contracted other diseases which had a more important bearing on the case. The connection shown by Dr. Gowers to exist between leucocythæmia and ague is also suggestive of a possible connection between the latter and splenic

* 'The Lancet,' vol. i, 1880, p. 173.

† 'Brit. Med. Jour.,' vol. i, 1884, p. 99.

‡ 'Diseases of Children,' vol. ii, p. 128. (New Sydenham Society.)

anæmia—a disease which, in many respects, seems to be allied to leucocythæmia. Such is the *a priori* reasoning in favour of ague being a possible cause of this splenic enlargement; but when we come to examine the cases individually, we find but little evidence of such an origin. Of the children which were in the hospital, some of them for many weeks, or even months, not one had a temperature chart in the least degree suggestive of intermittent fever, only a pyrexia associated with pulmonary inflammation or the irregular febrile attacks similar to those occurring in the course of other profound anæmias; not one showed any improvement under quinine or arsenic. But more interesting and decisive is it to contrast the clinical history of the cases already described with that of an undoubted instance of ague with enlarged spleen in a child. A boy, aged $2\frac{1}{2}$ years, was brought to me on June 19, 1891. He had been born in Mooltan, in the Punjaub. His father had been in India twenty years, and had had slight attacks of ague; his mother had had ague severely several times, both before and during her pregnancy with the patient. There was no suspicion of syphilis. When six months old the boy was seized with ague, and subsequently he had frequent and very definite attacks, with distinct cold, hot, and sweating stages. They had recurred since his arrival in England, a month before being brought to the hospital, and he had wasted considerably. When first seen, the child was distinctly anæmic, fairly well developed, and not rickety; had slightly enlarged glands in the neck, axillæ, and groins; the liver was felt a full inch below the costal margin, the spleen projected 2 inches below it, reaching forwards to the nipple line and upwards to the 7th space in the mid-axillary line; the blood showed 70 per cent. of red discs, about 1 white to 100 red, and 60 per cent. of hæmoglobin. Here, then, was a case in which the physical signs and symptoms were absolutely identical with those of a moderately severe case of splenic anæmia—pallor and some wasting, the same condition of the blood, moderate enlargement of the spleen, and slight hypertrophy of the liver and lymphatic glands. The only difference was that in this case there was a definite history of repeated attacks of ague. Here, therefore, was a patient in whom the reaction to treatment might decide whether the resemblance was completed by a similar chronicity and unresponsiveness to drugs to that which characterises true splenic anæmia, and the test was a very fair one too, for

the child must have been well under the malarial influence, not merely from exposure to infection after birth, but also by inheritance, in the most direct and powerful form, from the mother during intra-uterine life.

Here is the result: the child was given 1 grain of quinine three times a day; in a fortnight he seemed much better, and the spleen could only just be felt. In five weeks he had gained weight, and greatly improved in every way. The lymphatic glands were much as before, but neither the spleen nor the liver could be felt below the ribs; the red corpuscles had risen to 91 per cent., 1 white to 450 red; hæmoglobin, 65 per cent. A more complete contrast to the progress of an ordinary case of splenic anæmia, a more convincing proof that the latter cannot, at any rate usually, be dependent upon malaria, could hardly be desired.*

I turn now to the relationship to congenital syphilis, and here we have much stronger evidence of a definite connection. In eight cases the infants were undoubtedly syphilitic, in six there was doubtful evidence, and in sixteen no evidence of specific disease; but even in several of the latter the possibility of syphilis could not, of course, in out-patient practice be by any means absolutely excluded. So far as can be ascertained, seven of the first fourteen cases died, and only three of the sixteen apparently were free from specific taint. I think there is little doubt that the personal bias of the observer influences considerably, consciously or unconsciously, the percentage of cases in which, amongst a number of infants, hereditary syphilis will be diagnosed, some accepting as indisputable signs or evidence which others would either regard as worthless or would look upon with much doubt. Thus, if the observer start with a preconceived notion that these cases of splenic anæmia are essentially syphilitic, then he will probably find evidence, to him convincing, of syphilis in nearly every case, and will argue that as so many are proved to be syphilitic, any unexplained residue must be owing to insufficient

* This patient was seen again in February, 1892, after having left off all treatment for six months; he had had no more definite attacks of ague, but at times had not seemed very well; he was well nourished, but a little pale; the spleen was enlarged, though not so much as before, reaching nearly an inch and a half below the ribs. The case could not be followed out, owing to the return of the child to India.

knowledge of facts, which if known would probably confirm his view. If, like Parrot, we were to consider practically all rickets as due to specific taint, then, indeed, nearly every one of my cases would be syphilitic; so also, if cranial bossing or cranial tabes be regarded as sufficient proof in default of other signs, the percentage of cases of splenic anæmia due to syphilis would be at once increased; but this position has, I believe, been abandoned even by its original promoters, and my own clinical observations do not prompt me to make any attempt to rehabilitate the former faith. Necessarily, however, it must always be exceedingly difficult to say that a specific taint—insufficient it may be to lead to definite manifestations, but impossible to exclude absolutely—may not be at the bottom of an obscure disease like splenic anæmia. Apart, however, from all doubtful disputations, the fact remains that certainly a considerable percentage of these cases, and perhaps the worst among them, are associated with undoubted congenital syphilis; and the significance of the conjunction is not much diminished by the fact that in a large number of specific cases there is at no time any detectable enlargement of the spleen, although Cornil states that it is always hypertrophied. In the majority of hereditary syphilitic cases epiphysitis and interstitial keratitis are absent, yet no one doubts that they are essentially specific in origin. Often in examining syphilitic children I have found a slight splenic enlargement, the organ not feeling very hard, and there being no associated anæmia. This is doubtless comparable to the slight enlargement of the spleen described by Mr. Hutchinson in secondary syphilis, similar to that met with in the course of some acute specific diseases, and it usually soon disappears under treatment; but perhaps at times the slight swelling may develop into a more pronounced hypertrophy, and eventually into a well-marked case of splenic anæmia. Probably all intermediate conditions obtain, and are illustrated among the thirty cases. Dr. Gee, in a paper read before the Medical and Chirurgical Society in 1867, stated that the spleen is enlarged by simple hypertrophy, so that it can be felt during life in about half the cases of congenital syphilis, and that in about one-quarter of the cases the enlargement is really great, and that sometimes the liver and lymphatic glands are hypertrophied as well; that the majority of cases of great enlargement die, but that sometimes the spleen gets gradually smaller; also that occasionally an enlarged spleen may

be the only sign of an active syphilitic cachexia.* In Wilks and Moxon's 'Pathological Anatomy' it is stated that "in syphilis we often meet with hypertrophic enlargement of the spleen, no cirrhosis or lardaceous disease being present."

But whilst fully realising the very remarkable connection existing between congenital syphilis and enlarged spleen, we must admit that in a large number of cases, and many of them very severe, there is no evidence whatever of specific taint; cases, too, which run identically the same course and present the same pathological appearances as those associated with undoubted syphilis. But even more important is the result of treatment. The general rule in hereditary syphilis—at any rate as regards the earlier manifestations—is that the lesions either rapidly disappear under mercury, or else, if very severe and developed at an earlier period of life, soon progress to a fatal termination. But this is certainly not the case with hypertrophy of the spleen. The most active mercurial treatment, even in those cases in which the ordinary syphilitic manifestations are most pronounced, so far from being curative, appears to do harm rather than good so far as the splenic condition and the anæmia are concerned. Of course it may be replied that antisymphilitic remedies do not benefit locomotor ataxy due to syphilis, or quinine leucocythæmia started by ague; but these are very remote and indirect consequences of syphilis and ague respectively, whilst splenic enlargement, if due to syphilis, would surely have to be classed, from the period of its onset, &c., amongst the conditions usually very amenable to treatment. We conclude, therefore, that though there must be a very close connection between congenital syphilis and splenic anæmia, yet something further is required adequately to explain all the facts of the case, and that syphilis can be classed as a predisposing cause only.

Lastly, we have to consider the relation between rickets and splenic anæmia. In twenty-seven of my cases there was rickets, in four only slight, in the remainder more or less severe. At first sight these figures very strongly suggest that in rickets we have the true cause of splenic anæmia, and this was the view which Sir William Jenner adopted in his classical lectures on Rickets,†

* Dr. Colcott Fox and Dr. Ball ('Brit. Med. Jour.,' vol. i, 1892, p. 855) also found some enlargement of the spleen in 75 or 48·4 per cent. of 155 cases of inherited syphilis.

† Published in 1860 in the 'Medical Times and Gazette.'

in which he says that "albuminoid infiltration of such a degree as to cause very great enlargement of the spleen is rarely seen except in those of the rickety diathesis." Before, however, we accept this seemingly tempting view, there are certain difficulties to consider:—1. That in the very great majority of rickety children no enlargement of the spleen can at any time be made out. I believe all observers have been and are agreed on this point. Although, as I have already said in regard to syphilis, absence of invariable association does not disprove a causal connection, yet it at least suggests the probability of the action of some further cause, present only in a minority of rickety cases. 2. That there is no connection at all between the severity of the rickets and the size of the spleen or the degree of anæmia. The splenic hypertrophy may be very great and the bone changes very slight, or *vice versâ*. Jenner said that sometimes the bone disease is extreme and in others moderate or even trifling in degree. 3. In certain cases of splenic anæmia there is no evidence whatever of rickets. Case 7, in which rickets were absent, was first seen when the child was $2\frac{1}{2}$ years old, and, as she had only been suckled a short time, she might possibly have had a slight attack of rickets, all signs of which had disappeared; but the child (Case 17) which died at the age of only 3 months, with well-marked symptoms of splenic anæmia, had been fed exclusively at the breast; and Case 14, also, aged 7 months, had had no food except the mother's milk. Of course it may be said that even in these cases, despite their early age and the absence of improper feeding, rickets would have been found if carefully looked for, and can, in fact, only be excluded by examination and section of the bones *post mortem*. It is interesting, however, that even Jenner, after dwelling on the association of rickets with splenic hypertrophy, adds in a note:—"I have now under my care a child, aged 3 months only, whose spleen reaches below the level of the umbilicus. The child is suffering from catarrh. There are no signs of rickets or tubercle." And so experienced and careful a pathologist as the late Dr. Fagge, in opening the important discussion on rickets at the Pathological Society in 1880, said that he had found a large fleshy spleen in many children who had been free from rickets.

But some supporters of the rachitic theory adopt a slightly different line of argument. Thus Dr. Dickinson, at the discussion just referred to, said that "where the visceral change is most

marked, that in the skeleton is seldom extreme, as if the disease spent its force in one direction or the other ;” or practically, in other words, that enlargement of the spleen alone may almost be considered sufficient evidence of rickets. It is, however, somewhat unfortunate, albeit perhaps rather suggestive, that, *mutatis mutandis*, the same thing has been said of syphilis. Thus Dr. Gee, as already quoted, says that sometimes an enlarged spleen is the only sign of an active syphilitic cachexia. Surely, however, this is begging the whole question ; such a line of argument has the advantage of being necessarily unassailable directly ; but it seems to me, whether applied to syphilis or to rickets, to be as incapable of proof as it is of disproof. Both the statements quoted may be true at times, particularly, I think, as regards syphilis, if some family histories are to be trusted ; obviously both cannot always be true. Moreover, in some of my most extreme cases of splenic enlargement the rickety bone changes were very marked ; in fact, as already stated, I can trace no definite relationship at all between the two conditions.

The foregoing remarks open up a question of wide interest as to how far we are justified in diagnosing rickets in the absence of distinct bone changes. I realise most fully that rickets is not a mere disease of the bones, but is a nutritional failure, affecting profoundly every one of the tissues and organs of the body ; but, granting all this, may we not, ought we not to, admit that there may be conditions resulting from malnutrition in infants which are not necessarily rachitic—conditions which indicate a failure of nutrition along other lines than those producing the typical phenomena of rickets ? In other words, are we not endeavouring to simplify our classification at the risk of collecting several distinct morbid conditions under one heading ? True it is that under the rheumatic diathesis we have been able to bring together such apparently diverse conditions as arthritis, endo- and peri-carditis, chorea, pleurisy, tonsillitis, &c., to the great advantage of our pathology and treatment ; but, on the other hand, how many totally different functional conditions have been accumulated under that convenient refuge word hysteria, with very many unfortunate consequences ? That malnutrition may, and does, take on various forms is indicated by the fact that the most profoundly marasmic infants are seldom rickety. Should we not recognise more fully than perhaps we do at present that exposure to diverse

unhygienic conditions—bad air, insufficient light, defects of diet, either as a whole or as regards one or more of the important constituents, &c., acting in conjunction with different inherited tendencies or actual cachexiæ—may result in correspondingly diverse pathological conditions? In one case simple atrophy; in another, ordinary rachitis with tendency to excessive fatness; in another, rachitic bone-change with more or less wasting; in another marked nervous weakness, shown by laryngismus, tetany, facial irritability, or by actual convulsions—conditions, without doubt, closely allied to rachitic bone-change, but apparently bearing no very definite relationship to it, or finally, in some instances, leading to the condition with which we are now dealing—splenic anæmia. We know now that ordinary rickets is essentially due not only to improper feeding, but to a distinct kind of improper feeding. Is it not reasonable to suppose that other unhygienic conditions, acting alone or in combination, may have equally definite, though as yet not as clearly recognised, results? What the particular condition is which causes this splenic anæmia, whether it be inherited or acquired, whether it be dietetic or aerial, I know not. That it is closely allied to the syphilitic cachexia, and even more nearly related to the causes which produce ordinary rickets (perhaps because rickets is more common than hereditary syphilis), there can, I think, be no doubt; very probably without some such predisposing element it will not develop. That neither of these diseases alone is the efficient cause I am almost equally certain. Dr. Cheadle, in a paper on Rickets read before the British Medical Association in 1888, suggested that the cases of rickets in which enlargement of the liver and spleen is found are those modified by syphilis. No doubt as rickets predisposes to splenic anæmia, and as congenital syphilis does also, the combination of the two might be supposed to greatly augment this predisposition; but, on the other hand, if it be established that the disease can exist apart from rickets and also apart from syphilis, any theory which requires for its production the combination of the two can hardly be satisfactory.

The relationship of splenic anæmia to syphilis and to rickets is perhaps comparable to, though closer than, that existing between leucocythæmia and ague; we admit that ague may be a cause of leucocythæmia, but that it is so in only a minority of cases, and furthermore that only in a very small number of cases of ague—and

of very various degrees of severity—does leucocythæmia result, and that, therefore, some further cause must be found. So whilst admitting the influence of syphilis and of rickets in producing splenic anæmia, we need to seek some still more direct exciting cause.

The assumption of various well-recognised predisposing causes of splenic anæmia, requiring, however, some definite but at present unknown exciting cause for its development, would explain the fact that, whatever its apparent origin, the disease appears to run an independent course; that it presents, as seems now to be generally admitted, identical pathological appearances in children whether syphilitic, rickety, even malarial, or showing no other cachexia; and that, finally, special treatment has but little influence over it; so that even in the most notably specific cases, mercury seems useless, and that when associated with marked rickets the splenic hypertrophy and the anæmia improve much more slowly than the ordinary rachitic phenomena. If it be doubted whether any such single and definite cause as I have suggested be likely to be found, it may be remembered that until recently some dozen different causes of rickets were described, and it is only during the last few years, owing to the experiments of Mr. Bland Sutton at the Zoological Gardens, and the writings of Dr. Cheadle, that the disease has been attributed more and more definitely to deficiency of certain essential elements, notably of fat, in the diet.

One more interesting question arises which has a bearing on the possible cause of splenic anæmia—viz., as to its age incidence. Up to now I have described it as a disease of very early childhood. In my 30 cases the oldest is $2\frac{1}{2}$ years, and I have only been able to trace one case, No. 6, now aged $3\frac{1}{2}$ years, in which the disease has persisted much beyond infancy; but does it ever occur in later life? In Fagge's 'Medicine' it is stated to be "certainly rare." In the article on "Splenic Anæmia" in Keating's 'Encyclopædia,' it is said that the disease may occur in children of all ages, as well as in adults; but the only case referred to is one published in 1856 by Friedriech in a boy of $5\frac{3}{4}$ years. In the 'Pathological Anatomy' by Wilks and Moxon (third edition, p. 495) the subject is thus mentioned:—"There are other cases in which the spleen is enlarged to a great size, and this enlargement is the chief, if not the only, disease that can be discovered. Thus, Sir Spencer Wells

removed a spleen weighing 6 lbs., this being the only disease discoverable in the body, and the blood not being leukæmic; and Mr. Squire met with a spleen weighing 13 lbs., while the blood did not show a positive excess of white corpuscles. Similar cases have been met with of simple hypertrophy of the spleen without leukæmia, both in the living and in dead subjects. They are generally, but not always, at least during life, associated with anæmia, and so, indeed, as a rule, are all long-standing diseases of the spleen. These cases are the only ones that can be properly considered as pure examples of hypertrophy of the spleen; the cause of the enlargement is unknown, and the accompanying clinical phenomena need further investigation." I have met with one case in a boy of 7 years, which might be considered an example of splenic anæmia. He was admitted from my out-patients to the Victoria Hospital for Children, Chelsea, on February 6th, 1891. There was no evidence of congenital syphilis or of ague, and no history of acute specific disease since infancy. He had been ill three months, with increasing pallor and weakness. The boy was well nourished, but markedly anæmic; the liver reached 2 inches below the ribs, and the spleen below the level of the umbilicus; it was hard, smooth, not tender. A systolic murmur, probably hæmic, was heard all over the cardiac area; urine acid, no albumen. Red corpuscles 42·6 per cent.; hæmoglobin 30 per cent.; no excess of white discs, but it was difficult to distinguish the red from the white, there being apparently intermediate granular corpuscles. The boy had attacks of pyrexia lasting several days, during which the spleen got larger, and diminished in the inter-pyrexial periods. There were no hæmorrhages. The anæmia increased, and on March 18th the red corpuscles were 33·6 per cent., 1 white to 84 red; hæmoglobin 20 per cent. Neither arsenic nor quinine seemed to have the slightest effect. On account of the hospital being closed, the boy was sent to St. George's Hospital. His friends afterwards took him home, where he died, and unfortunately no necropsy was obtained. Except for there being no excess of white corpuscles, this case presented all the characters of leucocythæmia. Can it, and can the other cases to which reference has been made, be considered true examples of the splenic anæmia which occurs in infants? Perhaps others may be able to mention similar cases occurring after infancy. At any rate, we may, I think, safely

conclude that splenic anæmia is excessively rare after the first two or three years of life, that it occurs in fact during the period of incidence of rickets, and this would tend to increase the etiological relationship of the two diseases.

As regards treatment, I have but little to add to what has already been said by implication. Mercury, even in the most obviously syphilitic cases, has seemed quite useless. In Case 1 it was given both internally and externally, and after four months there was no improvement as regards the spleen, though ordinary syphilitic manifestations had disappeared. Mercurial inunction was tried for some weeks in Case 6, in which the spleen still remains enlarged; and also in Case 15, which proved fatal. In Case 17, for a month before death, mercurial ointment was applied and grey powder given by the mouth. The only patient in whom the spleen seemed to get smaller under mercury was Case 14, and this was a mild case from the first. Neither quinine nor arsenic had any apparent effect; thus in Case 4, liquor arsenicalis was given for five weeks in increasing doses up to 3 minims three times a day, and was eventually left off, owing to vomiting, without having caused any improvement. Also, in Case 8, 2 minims of liquor arsenicalis were given every four hours for some time without result. I should add, however, that in Case 5, a very severe one, the child was taken out of the hospital by the parents, after being in only a few days, and Mr. Mordaunt Wheeler, of Battersea Park Road, under whose care she then came, tells me that he treated her with increasing doses of bromide of potassium and liquor arsenici hydrochloricus for over twelve months, until complete recovery ensued. One great difficulty, especially in out-patient practice, in giving any of these remedies a fair trial, lies in the frequency with which the course of special treatment is interrupted by attacks of gastro-intestinal or respiratory catarrh—often protracted and severe—to which these anæmic children are particularly prone. As a rule, the most efficient treatment, in addition to suitable food, &c., appeared to be cod-liver oil and iron, and I am inclined to think that the latter especially may be pushed with advantage. Thus, in Case 8, one of the most severe of the series, the red corpuscles increased in six weeks from 37 to 63 per cent., and the hæmoglobin from 25 to 38 per cent. under reduced iron, given up to 8 grains a day. It should, however, be added that in this case the anæmia

Sex.	Age.	When first seen.	Nutrition.	Appearance.	Spleen.	Liver.	External lymphatic glands.	Rickets.	Cong. syph.	Blood (red corpuscles).	Result.
1 M.	12 months	March 22, 1889	Wasting	Marked anæmia	To iliac crest	...	All slightly enlarged	Moderate	Present	44 %	After 4 months in <i>statu quo</i> , not traced later.
2 F.	20 months	April 16, 1889	Always very thin	Marked anæmia and splenic tint	To iliac fossa	Reaches to 1" from umbilicus	All slightly enlarged	Marked	?	48 %	Not traced.
3 M.	2½ years	May 14, 1889	Not much wasted	No marked anæmia	Just felt below ribs	Edge not felt	All slightly enlarged	Marked	Absent	...	Great improvement after 7 months; spleen barely felt.
4 F.	13 months	July 23, 1889	Progressive wasting	Marked anæmia	Slightly enlarged	Distinctly felt below ribs	Inguinal slightly enlarged	Slight	Absent	44 %	Sudden death, Sept. 14, 1889. Spleen 1 oz., liver 15 ozs.
5 M.	13 months	Dec. 11, 1888	Not much wasted	Marked anæmia	To iliac crest	1" below ribs	Not enlarged	Marked	Absent	38 %	Nov., 1891—perfectly strong and healthy.
6 M.	18 months	Oct. 1, 1889	Wasted	Marked anæmia and splenic tint	To iliac crest	1" below ribs	Not enlarged	Moderate	Present	45 %	Oct., 1891—in Westminster Hospital in <i>statu quo</i> . Spleen slightly larger; red corpuscles about 50 % no excess of white.
7 F.	2½ years	Oct. 7, 1889	Well developed	Marked anæmia	To iliac crest	Edge midway between ribs and umbilicus	All slightly enlarged	Nil	Absent	64·5 %	Nov., 1891—strong and well; liver and spleen not felt.
8 M.	15 months	Dec. 27, 1889	Wasting	Moderate anæmia	To iliac crest	Edge just felt	All slightly enlarged	Marked	Present	64 % after epistaxis fell to 37 % and hæmoglobin 25 % after-wards rose again to 63 % and hæmoglobin 38 %	Spleen and glands increased in size; death from diphtheria April 24, 1890. Liver 12 ozs., spleen 4½ ozs.
9 M.	23 months	March 4, 1890	No marked wasting	No marked anæmia	Just felt below ribs	Edge not felt	Not enlarged	Marked	Absent	...	Sept., 1891—said to be quite well.

Analysis of Thirty Cases of Splenic Anæmia.

	Sex.	Age.	When first seen.	Nutrition.	Appearance.	Spleen.	Liver.	External lymphatic glands.	Rickets.	Cong. syph.	Blood (red corpuscles).	Result.
10	M.	5 months	Jan. 10, 1890	Fairly nourished	Marked anæmia	Distinctly felt below ribs	Edge not felt	Cervical and inguinal enlarged	Marked	Absent	42 %	Sept., 1891—quite well; liver and spleen not felt. Red corpuscles 104 %.
11	F.	2½ years	March 11, 1890	Not wasted	Fairly good colour	1½" below ribs	Edge midway between ribs and umbilicus	...	Moderate	Absent	75 %	Not traced.
12*	M.	12 months	May 1, 1890	Wasting	Marked anæmia	To iliac crest	Edge just felt	Not enlarged	Marked	Absent	58 % (hæmoglobin 30 %); after 16 days in hospital 60 % (hæmoglobin in 33 %)	Not traced.
13	M.	14 months	May 14, 1890	Fat, but said to have lost flesh	Marked anæmia	2½" below ribs	Edge distinctly felt below ribs	Not enlarged	Moderate	Present	72.5 % (hæmoglobin 34 %); 6 white to 725 red corpuscles	Nov., 1891—in good health; spleen 1½" and liver 1" below ribs; red corpuscles 90 %; 4 white to 900 red; hæmoglobin 50 %.
14	F.	7 months	June 24, 1890	Not wasted	No marked anæmia	Distinctly felt below ribs	Edge not felt	Cervical enlarged	Nil	Present	...	Sept., 1891—seems perfectly well; spleen not felt.
15†	M.	10 months	Sept. 4, 1890	Wasted	Marked anæmia	To iliac crest	1" below ribs	Cervical and inguinal enlarged	Marked	Present	32 % (hæmoglobin 30 %)	Died of pneumonia Sept. 22, 1890. Liver 10 ozs., spleen 5½ ozs.
16	M.	15 months	Sept. 23, 1890	Wasted	Marked anæmia	To iliac fossa	1½" below ribs	Inguinal enlarged	Slight	Present	...	Death Sept. 27, 1890. Liver 15½ ozs., spleen 8½ ozs.
17	F.	2 months	Nov. 11, 1890	Much wasted	Marked anæmia	To iliac crest	Edge midway between ribs and umbilicus	Not enlarged	Nil	Present	...	Death Dec. 11, 1890. Abscess of leg. P. M. refused.

* Father had ague when a boy.

† Father had ague twice.

Analysis of Thirty Cases of Splenic Anæmia.

IN YOUNG CHILDREN.

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	Sex.	Age.	When first seen.	Nutrition.	Appearance.	Spleen.	Liver.	External lymphatic glands.	Rickets.	Cong. syph.	Blood (red corpuscles).	Result.
18	M.	15 months	Sept. 26, 1890	Well nourished	No anæmia	1½" below ribs	1" below ribs	Inguinal and right axillary slightly enlarged	Slight	Absent	...	Oct., 1891—seems quite well; spleen can just be felt, liver and glands as before.
19	F.	18 months	Jan. 29, 1891	Wasted	Marked anæmia	1½" below ribs	Edge not felt	Cervical and inguinal slightly enlarged	Moderate	Absent	78% (hæmoglobin 26%); 1 white to 100 red corpuscles	Oct., 1891—still rickety; spleen 1" and liver 1" below ribs; red corpuscles 98%, 1 white to 245 red; hæmoglobin 45%.
20	F.	8 months	April 11, 1891	Slightly wasted	Slight anæmia	To iliac fossa	Edge midway between ribs and umbilicus	Inguinal enlarged	Moderate	?	62% (hæmoglobin 45%)	Oct., 1891—much fatter and looking very well; liver and spleen not felt; no enlarged glands; red corpuscles 82·6%; hæmoglobin 40%.
21	F.	13 months	April 28, 1891	Fairly nourished	No marked anæmia	1½" below ribs	Edge not felt	Not enlarged	Moderate	Absent	...	Nov., 1891 — much better; spleen not felt; good colour.
22	F.	11 months	May 19, 1891	Wasted	Marked anæmia	To iliac crest	1" below ribs	Cervical enlarged	Marked	Absent	70% (hæmoglobin 40%)	Child improved greatly and spleen got much smaller, only felt 1" below ribs. Died of whooping cough and pneumonia Aug. 23, 1891. No P. M.
23	M.	8 months	June 19, 1891	Much wasted	No marked anæmia	1½" below ribs	1¼" below ribs	Cervical slightly enlarged	Marked	Absent	...	Not traced.
24	F.	12 months	June 23, 1891	Slightly wasted	No marked anæmia	To iliac crest	Edge just felt	Enlarged, especially inguinal	Marked	?	...	Died suddenly July 10, 1891. No P. M.
25	M	12 months	July 14, 1891	Fairly nourished	No marked anæmia	1½" below ribs	Edge not felt	Inguinal slightly enlarged	Slight	?	...	Not traced.

Analysis of Thirty Cases of Splenic Anæmia.

	Sex.	Age.	When first seen.	Nutrition.	Appearance.	Spleen.	Liver.	External lymphatic glands.	Rickets.	Cong. syph.	Blood (red corpuscles).	Result.
26	M.	13 months	Oct. 25, 1888	Fairly nourished	Moderate anæmia	1½" below ribs	1" below ribs	Inguinal and cervical slightly enlarged	Moderate	?	...	Rickets rapidly increased; child died of broncho-pneumonia Nov. 20, 1888. Liver 12 ozs. Died June 8, 1889.
27	F.	18 months	May 23, 1889	Wasted	Moderate anæmia	Distinctly felt below ribs	Marked	Absent	...	Sept. 1891—child much improved; no anæmia; spleen barely felt.
28	F.	9 months	March 28, 1890	Slightly wasted	Slight anæmia	Distinctly felt below ribs	Edge not felt	Not enlarged	Moderate	Absent	...	Sept. 1891—general condition improved; spleen much as before.
29	F.	23 months	June 12, 1891	Fat	No marked anæmia	1½" below ribs	1½" below ribs	Cervical slightly enlarged	Moderate	Absent	...	Died of diphtheria Dec. 19, 1890. Liver 12½ ozs., spleen 4 ozs.
30	M.	15 months	Dec. 2, 1890	Much wasted	Marked anæmia	To iliac crest	2" below ribs	Not enlarged	Moderate	?	...	

had previously to the former estimation been unduly increased by severe epistaxis. Again, in Case 22, tincture of perchloride of iron was given up to 5 minims three times a day for several weeks, and during this time the spleen got very considerably smaller, and the child's colour and general nutrition greatly improved. Thus, in the future, whilst of course treating any obvious symptoms of syphilis, I shall be inclined to rely for the improvement of the splenic anæmia proper upon the, as far as possible continuous, administration of large and increasing doses of iron.

In this paper I have not brought forward new facts or advanced novel theories, but have simply endeavoured, by describing, and as far as possible following out, the history of thirty cases of varying degrees of severity, to arouse interest, to promote inquiry, and thus to increase our common knowledge of the origin, course, and so ultimately of the successful treatment of a disease of which at present we can only say that its pathology is obscure, its prognosis serious, and its treatment very unsatisfactory. I have to thank my colleagues at the Victoria Hospital, Dr. Ridge-Jones, Dr. Colcott Fox, and Dr. Drewitt, for allowing me to make use of their cases in the wards, and the house physician, Mr. Gale, for valuable help in the examination of the blood.

Dr. LESLIE OGILVIE said that in childhood an excess of hæmoglobin and of corpuscles was not infrequently met with. In cases of enlarged spleen the percentage of hæmoglobin was diminished and the corpuscles increased in number. The corpuscles showed a variety of form and size, and some were nucleated. In severe cases of rickets without splenic tumour the same condition was sometimes found. The excretion of urea was usually diminished, the urine being whey-like. As the child improved, the excess of corpuscles disappeared and the urea increased. He considered that syphilis was one of various debilitating influences which so alter general nutrition as to lead to rickets with or without splenic tumour.

Dr. COLCOTT FOX, alluding to the enlargement of the spleen in children, said that in three years he had collected sixty-eight cases, five of which he excluded as being tuberculous. In age the cases ranged from 4 months to 2½ years—that is, while the bone changes of rickets were progressing—and, as a matter of fact, the greater number were undoubtedly rickety. When one child was found affected in this way, succeeding ones in the family were apt to follow the example. He found anæmia usually well marked, the patients exhibiting a waxy, greenish tint, with flabby tissues, and commonly enlarged liver and lymphatic glands. In six cases in which the blood had been examined there was noted a diminution in red, and, probably, a slight increase of white, corpuscles. In the majority of cases the prognosis was favourable, but in a small number the enlargement of the spleen was progressive, and quite uncontrolled by drugs. Histologically, this appeared to be due to simple hypertrophy, with some increase of fibrous tissue. The changes in hereditary syphilis were apparently

identical. Recovery in the majority of cases took place under treatment with iron and cod-liver oil. In another series he had collected the cases of hereditary syphilis which came under his notice, of which 47 per cent. had enlarged spleen. He thought that a very large percentage of cases of hereditary syphilis ended in rickets, and that in cases of rickets with enlarged spleen it was often most difficult to exclude syphilis. He had found the spleen extending to the umbilicus in infants of a few weeks. His conclusion was that syphilis was a principal factor, aided by rickets, which might be an indirect outcome of syphilis.

The PRESIDENT inquired if any alteration had been noted in the urine with regard to the quantity of uric acid. He thought it would be interesting to know more about the association of marked anæmia with enlarged spleen, as to whether they were mutually dependent on one another, and also whether there were any abnormalities in connection with the red corpuscles such as occurred in pernicious anæmia.

Dr. CARR, in reply, said that he had estimated the corpuscles only in the most marked cases, and that a consideration of the whole number would probably somewhat increase the percentage. The association of the condition with rickets was not so very clear; this disease was absent in some, and syphilis in others, so that a third factor appeared necessary to explain it. Apart from intercurrent disease the prognosis appeared good. Systematic observations on the urine were practically impossible to carry out in infants, but so far as he had been able to note there was nothing special to remark upon. He thought the anæmia and the splenic enlargement progressed *pari passu*, but case No. 4 certainly seemed to be an exception to this.

NOTE ON THE TREATMENT OF SOME FORMS OF CHRONIC BRONCHITIS BY THE WATERS OF WEISSENBURG (SWITZERLAND).

By J. S. KESER, M.D., F.R.C.S.

WE are all familiar with those troublesome cases of winter cough where the symptoms reappear year after year, increasing each time in severity, and finally becoming permanent. We find at first the signs of simple bronchitis, and, later, see various complications, such as bronchiectasis, emphysema, &c. When the patients can spend part of the winter in a suitable climate, their condition often improves or remains stationary; those who are unable to leave the large towns at all, or only for a short time in summer, are most troublesome to deal with; I shall not here describe the various plans of treatment which are most frequently beneficial, but I will only remark that drugs often have a temporary effect only.

Even an ordinary change of air, to the seaside, or to the country, does not always produce a lasting amelioration ; and this has been very striking in a patient of mine who had been suffering from bronchitis for ten years ; she is 48 years old, strong, and very active ; her occupations oblige her to talk a good deal, and she is quite unable to leave London during the winter months.

For several years she had been under my care, and every summer there was a considerable amelioration in her condition, but never a complete disappearance of the symptoms ; she tried the seaside and country air in England and on the Continent, but as soon as she came back to London, and in spite of all precautions, the cough returned and increased gradually ; there was a moderate expectoration of frothy mucus or muco-pus ; on damp and foggy days dyspnœa set in, and sometimes became very troublesome ; râles were heard all over the chest, and the signs of bronchial catarrh persisted more or less until the next summer. In 1890 I decided to send her to Weissenburg, where she stayed for three weeks, and took the waters under the direction of Dr. Huguenin, formerly Professor of Medicine at the Zurich University. When my patient came back she had got entirely rid of her bronchial affection ; last winter she caught a slight cold, and coughed for about a week, but all symptoms promptly disappeared under ordinary treatment. This summer (1891) she again went to Weissenburg, and she is now quite well. She has had no cough during the last nine months, and there has been no dyspnœa. Careful examination of the chest reveals nothing abnormal, except slight emphysema.

My second case belongs to an entirely different class ; it is an example of that intractable and dangerous form of chronic bronchitis which is observed in phthisical patients ; this gentleman, 43 years of age, has on several occasions suffered from hæmoptysis ; the first attack occurred twelve years ago, and was very violent ; since then there have been five attacks, and when I saw the patient for the first time, eight years ago, he had well-marked signs of infiltration of the left apex. He got gradually better, and remained so for five years, when, in consequence of some imprudence, he brought up small quantities of blood ; bacilli were found in the sputum on several occasions. The cough persisted in spite of careful treatment, and never entirely disappeared until lately ; there were signs of widespread bronchitis, and on various

occasions the patient had to stay in bed for slight attacks of congestion of the lungs or blood spitting. In the spring of 1890 his condition was decidedly unsatisfactory; troublesome cough, abundant expectoration, dulness at the right apex, râles all over the chest, loss of appetite, and emaciation. During the summer 1890, the patient went to Weissenburg to undergo the usual course of treatment; when he came back he was so well that he was able to insure his life with a slight increase of the premium. He went to Weissenburg again last summer (1891), and when he came back nothing abnormal could be found except slightly prolonged expiration at the right apex. I saw him a few days ago, and there are no signs of bronchitis.

Weissenburg is situated in the narrow valley of the Bunschenbach, near Thoun, at a height of 874 metres; few places are so completely free from wind and dust as Weissenburg. The water contains in 1 litre 0.95 of sulphate of lime, 0.3 of sulphate of magnesia, and many other substances in small quantities; temperature at the "buvette" 22° to 25° C.; reaction slightly alkaline; no smell, and hardly any taste. From 150 to 800 grams of the water (previously warmed) are drunk every morning in three or four doses; it produces very abundant diuresis, and sometimes constipation. The patients are expected to follow strict rules concerning diet, amount of exercise, &c. Weissenburg enjoys considerable local reputation in the treatment of various affections of the chest, such as some forms of pulmonary phthisis, chronic pneumonia, pleurisy, and bronchitis. I am inclined to think that very good results would be obtained in cases similar to those I have described, and, as these forms of bronchitis are especially intractable in our London climate, an addition to the means we already possess may not prove altogether unprofitable.

Much has been written concerning the mode of action of the Weissenburg water; nothing certain is known on the subject except what might be explained by the increased diuresis; but it has been noted by competent observers that patients who had been staying at Weissenburg for a considerable length of time, only began to improve markedly after they had commenced to drink the water. Very likely several factors are at play: the almost absolute purity of the air, the large quantity of peroxide of hydrogen which it contains, the high degree of moisture, and the absence of wind are, no doubt, very important, although the

beneficial effects of the water are admitted by all those who have made a special study of the subject.

February 15th, 1892.

OBSERVATIONS ON THE CURE OR SUBSIDENCE OF ASCITES DUE TO HEPATIC DISEASE.

By JOHN S. BRISTOWE, M.D., LL.D., F.R.C.P., F.R.S.

THE subject of the subsidence of ascites dependent on portal obstruction, and the apparent restoration to health of persons thus affected and also suffering from permanent organic disease of the liver, is one that has interested me largely for many years, and is of great practical importance; for (notwithstanding that many experienced medical men know differently, and that the excellent papers on "The Varieties of Hepatic Cirrhosis," by Dr. Saundby, and on "Œsophageal Varices as a Cause of Hæmatemesis in Cirrhosis of the Liver," by Drs. T. Stacey Wilson and J. R. Ratcliffe, read before the British Medical Association in 1890, throw a very instructive side-light on the whole subject) I believe I am correct in saying that most medical men at the present time regard the development of ascites in connection with liver disease as of fatal omen, or, at any rate, the beginning of the end. In most cases I am free to admit that it is so, but it is certain that in no inconsiderable minority of such cases recovery takes place under suitable treatment.

I propose now to place before you several striking examples of recovery from ascites due to organic disease of the liver, and to add or interpolate such remarks in relation to them, and to the subject generally, as the cases suggest.

CASE 1.—H. H——, married, and a governess, about 44 years of age, was admitted into St. Thomas's Hospital under my care on August 8th, 1886. She had been a hard-working woman, had had much domestic worry, and for several years, though not getting drunk, had taken to drink. Her health had begun to suffer and her strength to fail about two years previously, and about six months before admission uneasiness or pain in the belly had come on, followed shortly afterwards by swelling of this part and in the legs. She had had diarrhoea for a few weeks, had

been liable to a cough for years, but had never observed that she was jaundiced.

She was a fat woman, with a slight but quite distinct icteric tinge. She had a cough unattended with expectoration, but there was more or less sibilant rhonchus over both lungs; the heart was healthy; the abdomen was large and somewhat tense, and contained some fluid, but there was no manifest enlargement of the liver, or tumour; the legs were œdematous; she was suffering from diarrhœa; the urine contained a little biliary pigment but no albumen. There was no further evidence of disease.

She remained under my treatment in the hospital from August 8th to May 24th, a period of nine months and a half. Although there was some variation in her symptoms, her health gradually deteriorated during the first seven months or so; she got progressively weaker, and a fatal issue seemed inevitable; then, after a short period of oscillation, she began to mend, her symptoms subsided one by one, she rapidly regained strength, and when she left the hospital she was stronger and better than she had been for years. The case is, of course, a long one, but its main facts may be epitomised in a comparatively few words.

Her mental condition for the greater part of the time she was under observation was peculiar; she had constant delusions and hallucinations; she saw children and cats moving about the ward; she saw persons getting in over the balcony; she heard the sister say her husband had been run over and killed, accusations made by the patients and others against her character, threats that she was to be taken downstairs and beaten; and she was generally low-spirited and often in tears. Her mental state began to improve about the beginning of February, and by the middle of the month had become normal.

During the whole period of her residence in the hospital she suffered more or less from bronchitic symptoms. Generally these were not of much moment, but occasionally she had a good deal of wheezing, with troublesome cough, attended with some muco-purulent expectoration. But there was never evidence of any organic lung disease; and the heart remained healthy, and for the most part its beats were not unduly rapid.

Her appetite was at first, and for a long time, in absolute abeyance; and she suffered a good deal from sickness and diarrhœa during the first month or two. She never, indeed, acquired a good appetite, and she suffered even to the last from occasional looseness of bowels. The motions always contained bile. The jaundice increased somewhat during the first two or three weeks, and was attended with the presence of bile pigment in the urine; but during the third month it gradually disappeared.

On admission, the abdomen was large, measuring $43\frac{1}{2}$ inches in girth, fat, and somewhat tense, and obviously contained fluid; and on August 21st was tapped for the first time. On that occasion only about 5 pints were removed. For a week or two there was no manifest change, but early in September it was noted that fluid was accumulating; and this process continued slowly, until on February 8th (or after a period of nearly six months) the abdomen had become very tense, and its girth had increased to $47\frac{1}{2}$ inches. She was then tapped for the second time, and 32 pints of serum were withdrawn. After the operation her circumference was found diminished by 10 inches. The ascites developed rapidly after this second tapping, and by March 11th she had become as large as ever, and on that day she had 34 pints of fluid removed, with an equal

reduction of bulk to that observed on the previous occasion. Again there was rapid re-accumulation, so that on the 22nd of the month (March) her girth was 43 inches, and on April 6th, 47 inches. But, although there had been this rapid development of ascites since her second tapping in February, her health in every other respect had been undergoing marked improvement; and from about April 6th, when for the third time her abdomen had attained its greatest degree of distension, improvement as regards the abdominal dropsy began also to take place. On April 14th her girth was $43\frac{1}{2}$ inches, on May 6th it was $39\frac{1}{2}$, and at the time of her discharge from the hospital only $33\frac{1}{2}$, and all evidences of ascites had disappeared. The great diminution in the size of her abdomen at this time compared with its diminution after her tapplings was doubtless due to the fact that, in addition to the removal of fluid, there had been a considerable removal of fat. During the whole of the time she was under observation the liver could never be felt, and there were no enlarged veins visible in the abdominal walls. The urine was always free from albumen.

At the beginning of May she had an attack of rheumatism (or gout), in which some of the small and several of the larger joints were successively affected. For this she was treated with salicylates, and recovered in the course of a fortnight.

Her temperature during her residence in the hospital rarely reached 100° , and was generally normal or subnormal; it became somewhat elevated, however, at the beginning of the rheumatic attack, and on one occasion rose to 102.8° .

As before stated, the patient left the hospital apparently in unusually good health on May 24th, 1887; and she continued in excellent health and capable of performing all her duties for the next two years. I used often to hear of her, and occasionally to see her, during all this period, and have reason to believe that for a large portion of it she either abstained wholly from alcohol or partook of it very sparingly, but that latterly she resumed her evil habit; and on August 29th, 1889, she was again admitted into the hospital under my care.

She had then been ailing for three months, mainly from gradual enlargement of the belly, but also from gradually increasing muscular debility.

On admission she was well nourished, of sallow complexion, but not jaundiced, and had no œdema of the legs. She had some cough, and wheezing was heard all over the chest, but she was not specially short-breathed. The abdomen was large and tense. Her legs were weak, and she had muscular hyperæsthesia with absence of knee-jerks; but there was no definite paralysis or loss of feeling. She did not now suffer, as she had done on the former occasion, from sickness or diarrhoea, or from mental disturbance, with the exception that there was some loss of memory. Her tongue was clean, her appetite fair, her urine free from albumen, her pulse about 80, and her temperature normal.

She remained in the hospital on this occasion from August 29th to January 12th, 1890, a period of about four months and a half.

The day after admission, 18 pints of clear straw-coloured fluid were removed from the abdomen. The muscular weakness which was observed at this time, more particularly in the legs, was the first indication of the coming on of peripheral neuritis. This affection increased upon her for two or three weeks, and was characterised by numbness and a sense of tingling and grittiness in the fingers and palms of the hands, with some

tenderness of these parts, and in the arms, and with some tremor, but no marked paralysis; and, as regards the lower extremities, by numbness and tingling in the feet and lower part of the legs, tenderness in the calves and feet, loss of power which prevented her from standing and walking, but not from moving her toes or feet while lying in bed, and abolition of knee-jerks. Then improvement gradually took place, and after she had been in hospital three months, she began to get up and walk about a little. Excepting that her knee-jerks had not returned, all her paralytic symptoms had disappeared by the time she had left the hospital. There was never any obvious wasting of the muscles of the affected limbs. During the earlier part of her stay in the hospital she had slight swelling and pain in the knee-joints, and during the whole of the time more or less of the bronchitis to which she was liable.

After the paracentesis, performed the day after admission, she gradually refilled, and on October 4th, by which date her girth was $43\frac{3}{4}$ inches, she was again tapped, and 20 pints of fluid were taken away. After this she again filled, until on December 13th her girth had attained 42 inches. From this date the dropsy gradually subsided; and a few days before her discharge the abdomen, though still containing a little fluid, measured only 39 inches. No enlargement of the liver or spleen was ever detected; and when she left the hospital on January 12th, 1890, she was fairly well.

Owing to the various symptoms she presented and her many complications, she was subjected while in the hospital to much variety of treatment; but the aim always in view was to treat her with tonics, to feed her well, and to withhold alcohol.

I have seen her occasionally since, the last time being one day last week, when she brought her daughter to consult me; and, excepting that she continued to suffer more or less from slight chronic bronchial catarrh, she has remained quite well, and there has never been any further development of dropsy. I am not sure that she does not occasionally drink even now, but I know that her family keep a very close watch over her.

That the case just related was a typical case of chronic alcoholism there can be no reasonable doubt; her history, her mental condition during her first spell of illness, and her neuritis during her second spell, all support this view. And it may therefore be accepted, I think, that the ascites was due to cirrhosis of the liver, and that (though she seems well) her liver is still cirrhotic.

CASE 2.—A gentleman, with whom I was formerly well acquainted, had a wife who for the last ten years of her life suffered from hysterical paraplegia, and became also irritable and exacting—a combination which made his home miserable, and led him to neglect his family, and to become immoral and intemperate in his habits. In the latter part of 1878—at which time he was 35 years of age—his wife died in her confinement. He at once deserted his house and children, leaving the latter dependent on

his relatives, and went into cohabitation with a woman of loose character with whom he had been familiar for some time, and whose acquaintance he had made at some refreshment bar in the City. Shortly afterwards he married her, and I lost sight of him until October, 1882, when I received information that he was lying very ill at a public-house, kept nominally by himself, but really by his wife. I called upon him on October 8th, and then learnt that he had been drinking very heavily ever since his first wife's death; that during the previous six months he had two or three times been on the verge of delirium tremens; that for some time he had been suffering from constant sickness, and getting weak and thin; that about three weeks ago his legs had begun to swell, and, a little later, enlargement of the belly had been observed; but that under medical treatment the sickness had now subsided, and his appetite was returning. I found him very ill, very thin and weak, and with a good deal of fluid in the abdominal cavity and in the lower extremities. I saw him two or three times during the following few weeks, and on November 20th made the following note:—

“Is getting much worse; is very thin and weak, and irritable, and constantly rambling. Takes little food, but is not sick; tongue clean, bowels regular. He is short-breathed; his pulse is weak and rapid. The abdomen does not appear to have enlarged materially, but it contains abundant fluid. There is also some fluid in the left pleural cavity.”

On November 22nd he was removed to St. Thomas's Home, and on November 24th I saw him there. He was then very ill, slightly jaundiced, and suffering much from dyspnoea. The abdomen contained, as before, a good deal of fluid, but the left pleura was now distended. Paracentesis thoracis was performed, and 3 pints of serum were removed, with much relief to the patient. A month later, namely, on December 22nd, I made the following note:—

“He has been improving in all respects. He has been steadily gaining flesh, strength, and appetite. His jaundice has disappeared. There has been no re-accumulation of fluid in the chest; the ascites has almost wholly subsided, and he has no oedema of the legs. He is still, however, very thin and weak, and has the aspect of a person suffering from grave organic disease of the liver.”

He continued to improve, and, excepting that he remained weak and extremely thin, he left fairly well on January 24th, 1883. I should add that he had no trace of cardiac, renal, or other visceral disease.

During his residence in the home his wife disappeared; and, shortly after leaving, his friends (who retained the charge of his children) helped him to emigrate to New Zealand. And there he has remained ever since, leading a lonely life, earning a scanty pittance by the sweat of his brow, but continuing physically in good health. As to his habits I know nothing, but assume that if he drinks at all, as probably he does, he drinks in moderation. There is no record of this patient's treatment, but I know that the treatment relied on was such as was calculated to give appetite and promote strength.

Now here again there was a very clear alcoholic history; and although the ascites got well without tapping, there can be no doubt, from the fact of the development of this form of dropsy and of jaundice, that his liver was at that time affected with early cirrhosis.

CASE 3.—On October 24th, 1888, I saw, with Dr. Purkiss, of Brentford, an eating-house keeper, about 40 years of age. He was a highly respectable, energetic, and successful business man, but for a long time had been in the habit of taking many glasses of spirits daily. He had generally had good health, but for some months his complexion had been getting sallow. He considered, however, that he had been ailing only for a month or six weeks, during which time his belly had been getting large and his legs œdematous. Nevertheless, he had eaten and slept well, and had felt well.

At the time of my visit, he was distinctly, though slightly, jaundiced; his liver was much enlarged, his abdomen was distended with fluid, and there was some œdema of the legs; but in all other respects he appeared to be sound.

He was, of course, enjoined to give up alcohol; he was ordered a tonic, and also to take a pill of mercury, squill, and digitalis two or three times a day. It was further arranged that he should be tapped.

I received a note from his doctor about two months later (namely, on December 28th), from which the following is a quotation:—"I have now tapped our patient five times (namely, on October 25th, November 5th, November 19th, December 3rd, and December 20th, on which occasions 9, 13½, 19½, 19, and 15 pints of fluid respectively were drawn off). I think his liver has now slightly diminished in size; and although he lost flesh considerably for the first four or five weeks after your visit, he has now regained flesh slightly. His urine still contains bile and lithates, but at times has been fairly clear; he takes nourishment pretty well. He has been taking the pills twice a day, and a mixture containing chloride of ammonium, spirits of nitrous ether, and gentian." Though I never saw this patient again, I have had frequent talks with my friend about him, for I was much interested in the progress of his case; and recently I have been favoured with the following brief narrative of his progress since December 28th, 1888.

Subsequently to the last recorded tapping on December 20th, 1888, he was tapped on February 8th, 1889, June 26th, July 30th, and August 19th, the quantity of fluid removed on each occasion varying from 8 to 16 pints. During the greater part of this time, he was taking quinine three times a day, and the diuretic pill.

At the end of August he went to Vichy, where he remained about three weeks. Whilst there he drank the waters, but took no medicine; and was tapped once. Towards the end of his visit he caught cold, which was aggravated while crossing the Channel on his homeward journey.

When seen by Dr. Purkiss the next day (September 22nd) he was found to be suffering from bronchitis and pleurisy. This illness was a serious one, and for a time a fatal issue was feared; but it was recovered from at the end of two months.

He was next tapped on February 5th, 1890, and then on March 3rd. In April he went to Dublin for eight or ten days to consult Dr. Hamilton, who again tapped him, and gave him a prescription for a mixture containing $\frac{1}{16}$ th gr. of iodine in each dose. This he has continued to take, off and on, ever since. On July 29th he was tapped for the last time, and appears to have had good health ever since.

Dr. Purkiss (in consequence of a letter from me) visited the patient on the 8th of this month, and reports as follows:—"I have seen him to-day, and find him in excellent health and spirits; he carries on his business with his old energy, generally gets up about 9 o'clock, and works from

that time till 11 P.M. He goes to market three times a week. His abdomen causes him no inconvenience, his bowels are regular, the appetite good. I am sorry to say he still takes some gin and water."

Altogether the patient was tapped fourteen times ; the first time being about three years and four months, the last more than eighteen months, ago.

In this case, as in the other two, the alcoholic history is indisputable, and that the ascites depended on hepatic disease is shown both by the enlargement of the organ and by the temporary jaundice. The next case belongs to a different category.

CASE 4.—On April 30th, 1890, I saw, with Dr. Wyman, a young lady about 24 years of age. She belonged to a highly respectable and, apparently, healthy family ; and, although of small and delicate physique, seems to have had good health down to the commencement of her present illness. About the immediately preceding Christmas, as she was about returning home from a foreign country, where she had been on a visit, she was suddenly attacked with profuse hæmatemesis and melæna. This delayed her return, but she arrived at a seaport in England, fairly well, about a week before I was called in. There she had a recurrence ; but, notwithstanding, was brought home in the course of a day or two, and there had two other attacks, the last being on the evening of the 29th. She had suffered latterly from pain or uneasiness after food and loss of appetite ; but (apart from the bleeding) the symptoms referable to the stomach had never been severe.

She was extremely anæmic and weak when I saw her, and complained of noises in her ears, but was otherwise comfortable and free from pain ; and, after careful examination, I failed to detect any evidence of thoracic disease, or any tumour or tenderness in the abdomen. We not unnaturally regarded the case as one of simple ulcer of the stomach, and treated her accordingly.

During the following ten or twelve days she progressively improved, had no return of bleeding, and at the end of this time was beginning to take a moderate quantity of appropriate food without much discomfort ; but, of course, she still remained pale and weak. Then the abdomen began to fill, and it filled so rapidly that within two or three days of the discovery of this condition, namely, on May 15th, she had to be tapped, and 11 pints of clear limpid serum were removed. I saw her on the 18th, three days after the operation, and already the abdomen was distended. I need scarcely say that the original diagnosis had now to be rejected, and that her symptoms were referred to portal obstruction, presumably dependent on some form of cirrhosis. But there was no reason to suspect her of over-indulgence in alcohol.

The next time I visited her was on May 28th. She had been tapped three times since my last visit, and each time to about 9 pints ; the last occasion being the day before the present interview. She looked much better in health than before, and was cheerful. She had a fair appetite, no sickness, and very little uneasiness after food. There was very little water in the abdomen at this time. The part was not tender, and, on careful examination, no enlarged organ or tumour could be felt. She was passing rather a large quantity of urine.

I never saw her again, but I heard a good deal about her from time to time, and discussed with Dr. Wyman the pathology of the case and its treatment.

Shortly after my last visit a consulting surgeon was called in, who himself performed paracentesis, and took some of the ascitic fluid away with him for examination; and either then, or subsequently, the edge of the liver was detected extending a little below the ribs, and in it a small nodule or area of hardness. The diagnosis then formed was, I believe, that the patient was suffering from malignant disease in the abdomen.

During June she was tapped seven times, at intervals ranging from three to seven days, the quantity of fluid varying between 4 pints 15 ounces and 8 pints 10 ounces. At the end of this month, however, a very suggestive item of information came to light. It was ascertained accidentally that a brother of this young lady had, a few years previously, been under treatment by a London oculist for some congenital syphilitic affection of the eyes, and naturally at once the suspicion was raised that the lump which had been detected in her liver was a gumma, and that the portal obstruction which had caused gastro-intestinal hæmorrhage and ascites was due to some growth of the same nature. 10 grains of iodide of potassium, to be taken three times a day, were therefore ordered for her early in July, and after two or three weeks the dose of iodide was increased to 12 grains, and a drachm of the solution of perchloride of mercury was added; and this treatment was continued, with a few interruptions, down to February in the following year.

During July (as during June) she was tapped seven times, to quantities varying between 5 pints 9 ounces and 8 pints 10 ounces; and during August six times, to quantities ranging from 4 pints 12 ounces to 7 pints 6 ounces. In September she was tapped three times, namely, on the 5th to 6 pints 17 ounces, on the 13th to 6 pints 10 ounces, and on the 22nd to 6 pints. This, which was the twenty-seventh operation, was the last that was ever needed. The patient thenceforth gradually regained fair health, and is, I am told, at the present time better than she has been for years.

In the case just narrated (although at first the symptoms were misleading and their cause obscure) it was eventually conclusively shown by the collateral history, by the presence of a nodule in the liver, and by the result of treatment, that the obstruction of the portal vein which had caused hæmatemesis and ascites was due to the growth of gummata about the transverse fissure of the liver.

I may add here that a case very similar to the last has been recorded, I think by Dr. Grainger Stewart, and that I can recall more than one case in which identical symptoms have resulted from cancer of the lesser omentum, or in the transverse fissure of the liver.

In conclusion, I propose to make a few remarks on the chief causes of obstruction to the circulation through the liver which lead to ascites, on the pathological or physiological means by

which this obstruction becomes obviated, and on the treatment of the kinds of cases under consideration.

Without going into minute particulars, we may classify the causes of obstruction as threefold: first of all, true cirrhosis, and other cirrhotic conditions of the liver, in which the seat of obstruction is the smaller and capillary vessels generally throughout the organ; secondly, cancerous and syphilitic growths occupying the transverse fissure of the liver or its neighbourhood, and compressing or involving the portal vein itself, or the occlusion of this vessel by the formation of thrombi; and thirdly, obstructive disease of the heart or lungs inducing nutmeg liver, and (owing to the relatively feeble force with which the blood returns from the chylipoietic viscera to the vena cava) relatively greater sluggishness in the flow of blood in the veins and capillaries of these parts, and therefore relatively greater tendency to dropsy in the abdomen, than elsewhere. My first three cases obviously belong to the first class, and my fourth case to the second class. Of the third class I have adduced no examples, partly because I should thereby have added considerably to the length of my paper, and partly because I have made it the subject of a clinical lecture which I am intending to publish.

The pathological means by which the portal obstruction becomes obviated, or cured, is a subject of much interest. In the first place, it has been amply proved that there are fairly free communications between the tributaries of the portal vein and neighbouring systemic veins; and that hence, when portal obstruction exists, there is a tendency for the blood of the portal circulation to be shunted into some of these other veins, which then gradually undergo dilatation, and thus for the more or less complete relief of this circulation. So that even without any beneficial change in the liver itself, ascites may in this way be permanently cured or circumvented. It has been shown (and very conclusively shown in the paper by Drs. Wilson and Ratcliffe, already referred to) that the œsophageal veins are largely concerned in this process; and that the consequent varicose condition of the submucous veins of this part is, from their tendency to rupture, one of the sources, if not the main source, of the profuse hæmatemesis and melæna which are apt to follow on portal obstruction. I have no doubt of the frequency of gastro-intestinal hæmorrhage from this cause; but I cannot help suspecting that, while this would be likely to

occur mainly in the later stages, the hæmorrhage attending the disease in its earlier stages may be due to the dilatation and rupture of some of the primarily over-distended and dilated submucous veins of the stomach or intestines. At any rate such perforations would be very difficult of discovery. But we cannot refer all cases of apparent recovery to this diversion of the portal circulation; for in many cases of recovery we have no evidence whatever that anastomosing veins have become dilated; and, moreover, if that were the universal explanation, cases like my first, in which after a time the dropsy subsides, to be redeveloped several years later after the lapse into bad habits, would be inexplicable. Indeed one cannot reasonably doubt, that in a slowly progressive inflammatory affection like cirrhosis, the progressive increase of which is in many, if not in most, cases due to the repeated influence of an irritant locally applied, the cessation of this influence would be likely to be followed not only by arrest of the disease, but also by some amelioration in so much of it as had recently accrued and was amenable to treatment; and further that, inasmuch as in cirrhosis there is a point in the progress of the disease at which dropsy does not arise, and a point, only a little later, at which dropsy does arise, by proper treatment at the right time this last straw that breaks the camel's back might be removed. It is quite clear that in each of my first three cases something of this kind occurred; for in each of them there was jaundice in relation with the beginning of ascites, a condition which implied a wider diffusion or a greater intensity of disease than that causing only dropsy, and which subsided. Again, there are other cases in which, as in my fourth, the obstructing disease is of a specific kind, and admits of amelioration or removal by specific treatment.

The question of treatment is an important one, yet one about which I will not discourse at any length. It is almost needless to expatiate on the value of accurate diagnosis in relation to this matter. My fourth case is a striking illustration of this fact, for it was one that would doubtless have ended fatally had it not been for the accidental discovery that a brother had been the victim of congenital syphilis. There are doubtless many cases of visceral syphilis which are misinterpreted to the detriment of the patient. The treatment of ascites due to cirrhosis of the liver is, according to my experience, comparatively simple, and in a large pro-

portion of cases proves very fairly successful. It is simply to promote the general health of the patient by appropriate tonics and diet, to cut off, as far as possible, the alcohol which he has probably been in the habit of taking, and to tap from time to time, not, however, waiting until the abdominal distension has become so great as of itself to impair health. Of course the tonic and dietetic treatment will need to be varied in different cases, and from time to time in the same case, in accordance with the condition of the digestive organs and the different symptoms or complications which may arise; and, further, such symptoms or complications may need specific treatment for their own amelioration or cure. But, although I should certainly keep the bowels freely open, I have never seen any benefit from active purging, and should oppose it. Indeed, spontaneous diarrhœa is not an uncommon incident in cases that are going on badly. Diuretics are of more service, and I have often thought that the combination of mercury, fresh squill, and digitalis has aided the removal or absorption of fluid. But they only act vicariously (so to speak) of paracentesis, and, though often beneficial, are not curative in the true sense of the word. Diaphoretics will, of course, act somewhat in the same way as diuretics, but I have never seen any obvious advantage from their employment. I should not, on theoretical or other grounds, object to the use of mercury or iodide of potassium in the treatment of the disease in its early stage.

The PRESIDENT asked Dr. Bristowe's consideration of a point with regard to his first two or three cases, viz., the presence of œdema of the legs as an *early symptom* in association with ascites. The question occurred to him whether in these cases the ascites undoubtedly due to cirrhotic liver was not precipitated by a paretic state of vessels and heart due to alcoholism. It was a common clinical experience that, given a general cause of dropsy, *e.g.*, a dilated or diseased heart in association with a cirrhotic liver, peritoneal dropsy becomes a disproportionately prominent symptom, and he desired to raise the question whether the earlier attacks of ascites in Dr. Bristowe's cases were not in part due to alcoholic changes in the vessels and to weakened heart. Œdema of the extremities was not, in his experience, an early feature in association with ordinary ascites.

Dr. DE HAVILLAND HALL pointed out that in some cases repeated tapings set up peritonitis, and that by the adhesions formed between the intestines and the abdominal walls, an increase in the collateral circulation was established, and that this was sometimes sufficient to cure the ascites. He mentioned a case of the sort which had come under his own observation. As regards the medicinal treatment of ascites, he thought that great benefit resulted from ringing the changes in diuretics. He had found the

resin of copaiba in 10-grain doses, as recommended by Dr. Wilks, of great service in stimulating the kidneys to act, but at times the resin seemed to upset the stomach.

Dr. HADDEN had made a *post-mortem* examination on a man who had been under the care of the late Dr. Murchison twelve or thirteen years previously for cirrhosis of the liver. The liver was found in an advanced stage of cirrhosis ; but death had occurred independently of this disease.

Mr. SHEILD dwelt on the importance of scrupulous care in tapping in cases of ascites. The instrument should be a clean aspirator, and the needle employed a small one. Many of these cases died from a low form of peritonitis, the result of tapping. He asked Dr. Bristowe whether in his third case the symptoms may not have been due to thrombosis of the portal vein, the result of gummatous phlebitis. He concluded by dwelling upon the great importance of examining the choroid in cases of suspected congenital syphilis, and asked Dr. Bristowe to explain cases of cirrhosis which occurred in very young children, without evidence of alcoholism.

Sir HUGH BEEVOR quoted two cases of marked alcoholism long continued, in which, without ascites being present, it was presumed from other symptoms that there was cirrhosis of the liver. One of these, a woman, had marked œdema of the legs ; there was no evidence of any anæmia, weakness of the circulation, or renal disease ; the œdema was attributed to the alcoholism or cirrhosis. The other case presented over the hepatic region many varicose venules of a diameter as great as that of an ordinary pin. He inquired if in any way these could be taken as evidence of collateral circulation.

Dr. SIDNEY PHILLIPS was interested in the case Dr. Bristowe mentioned which recovered under treatment by potassic iodide ; he thought that cases of ascites which were not of syphilitic origin were also sometimes cleared up by iodide of potassium ; he had seen three cases in which no specific history was obtainable, and in which there was nothing to point to syphilis, which cleared up with iodide of potassium, but the drug required giving in larger doses than were usually ordered. Dr. Phillips asked Dr. Bristowe for information respecting ascites in children—not tubercular, which got well with rest and tonics.

Dr. PASTEUR observed that ascites was far from rare amongst children, and that such cases, in his experience, mostly tended towards recovery under appropriate treatment, often without recourse to tapping. Some of these cases are obviously tubercular ; but in others, where the ascites is practically the only evidence of disease, there may be great difficulty in distinguishing a tubercular effusion from one due to portal obstruction. In his experience, however, ascites from hepatic cirrhosis was a very rare event in childhood, whereas ascites due to the presence of tubercle in the peritoneum was relatively common, and in not a few instances appeared to constitute the only evidence of tuberculosis.

Dr. BRISTOWE, in reply, said he regarded the œdema of the legs so common in connection with ascites as due to the collection of fluid in the pelvis and pressure on veins there. He mentioned other cases similar to those quoted, but of which the notes were less complete. He thought the examination of the eyes was of great value in recognising syphilis, but said that the signs were sometimes so slight as to have been overlooked even by skilled observers. In young children tubercle, alcoholic cirrhosis, and congenital syphilis had to be considered in relation to the causation of ascites.

HÆMATEMESIS, WITH SPECIAL REFERENCE TO THAT FORM MET WITH IN EARLY ADULT FEMALE LIFE.

By DONALD W. CHARLES HOOD, M.D. Cantab., F.R.C.P.

IT is my intention to bring before the members of the Medical Society of London those clinical features which I believe will generally be found accompanying this form of hæmorrhage, and which I believe tend to differentiate it from hæmorrhage arising from organic disease of the stomach.

I will endeavour to show that these attacks of hæmorrhage—so often preceded by profound blood changes of anæmic type—are not necessarily due to ulceration, but that the balance of clinical and pathological evidence rather suggests the hæmorrhage as being of a more simple nature, possibly due to derangement of circulation fundamentally produced by the underlying anæmic condition.

Hæmorrhage from the stomach is a factor which has always occupied a position of extreme importance in weighing the value of symptoms referable to the stomach. With many—I might say with almost all—it has been the one symptom which is looked upon as conclusive in the differential diagnosis between organic and functional disease of the stomach, between the various forms of dyspepsia and ulceration of that viscus.

If the pages of Brinton and Habershon be consulted, it will be found that both these observers state that the diagnosis of ulcer can be made in but very few instances, unless this symptom, hæmorrhage, be present. I will not take up the time of the Society by mentioning more than the remarks made by these two physicians on these points; practically, their views are quoted again and again through the various text-books on general medicine.

If we consult Brinton's work 'On Ulcer of the Stomach,' on p. 104 will be found the following words—he is writing on the differential diagnosis of stomach ulceration: "Unless the pain possess the characters attributed to it, unless this pain be accompanied by vomiting, and *unless* there be evidence of hæmorrhage

having occurred in the course of the malady, there is no sufficient basis for a definite diagnosis of the existence of gastric ulcer."

Speaking generally, this opinion of Brinton will be found more or less to influence the diagnosis of gastric ulcer at the present day, and the later authority on diseases of the stomach, namely, Dr. Habershon, expresses his opinion quite as strongly. He writes: "Unless hæmorrhage, however, take place, we cannot with any certainty diagnose ulceration of the stomach."* He is in perfect accord with Brinton, that hæmorrhage is the one symptom which differentiates the various forms of gastric disturbance from that caused by ulceration.

My own observations would lead me to think that the value of the symptom is in many cases overrated. It is a symptom undoubtedly present in a very large number of cases where organic disease of the stomach has been proved to be absent; and again, a matter of extreme practical importance, it is that pathognomonic symptom which is absent in numbers of cases where ulceration is eventually found to be the cause of stomach trouble.

Apart from my own personal experience, which relates to about forty cases of hæmatemesis occurring among females, I have been anxious to obtain reliable data to assist me in my investigation as to the proper value to be awarded to the symptom.

Dr. Goodall, lately medical registrar to Guy's Hospital, has very kindly with great care examined for me the clinical and pathological record of Guy's Hospital, during a period of twenty years, 1870-90. I should add that Dr. Goodall informs me that all the cases to which reference will be made are undoubted. The hæmorrhage occurred while the patient was under observation, and in no instance was the mere statement of the patient taken as sole evidence.

These cases (155 in number) will be considered under the following heads:—1. Cases where the hæmorrhage did not prove fatal, but where the patient was discharged from the hospital as "well" or "relieved." 2. Cases where the patients died from the direct effect of the hæmorrhage. 3. Cases where the hæmorrhage did not prove immediately fatal, but where the patient died at some later date while under observation.

Under the first head—non-fatal cases—there are 118 patients.

* 'Diseases of Abdomen,' third edition, p. 183.

Of these, 59 were males and 59 females. They were diagnosed as follows:—66 cases of simple gastric ulcer, 19 cases of hæmatemesis only, 16 cases of cirrhosis, 5 cases of gastritis, 4 cases of carcinoma, 2 cases of chronic Bright's disease, 2 cases of injury, 5 cases doubtful.

Nineteen patients died from the direct effect of the hæmorrhage. Of these 11 were men and 8 women.

In 9 of these cases death was due to cirrhosis, in 7 to ulceration extending into a large vessel, and in 3 to the rupture of an aneurism. 18 patients—14 men and 4 women—suffered from severe hæmorrhage not fatal at the time. Of these cases 7 were the subjects of cirrhosis. An equal number had malignant disease. With 2 patients ulceration of the stomach complicated other diseases, (*a*) granular kidney, (*b*) phthisis. Two patients suffered respectively—the one from cardiac disease, the other from peritonitis following perforation.

In estimating the correct value of the symptom, hæmorrhage, it is necessary to take into consideration the age of the patient. Even at the risk of being wearisome, I must ask the attention of the Society on this point.

Among the 118 patients discharged from Guy's Hospital as cured or relieved, there were 36 cases under 30 years of age, 27 being females and 9 males. Beyond 30 years of age there were 82 cases—51 males and 31 females. Among those fatal cases in which death immediately followed the perforation of a blood vessel—7 in number—there were 4 females, aged respectively 33, 35, 50, 53; 3 males, aged 37, 43, 60. Nine deaths followed the hæmorrhage associated with cirrhosis—6 males, aged 17, 18, 36, 40, 42, 47; 3 females, 28, 46, 54. In 18 cases (14 men and 4 women), although not directly fatal, the hæmorrhage had been a serious symptom before death. Among men, 3 were the subjects of a perforating gastric ulcer, their ages being 23, 57, 62; 6 were the subjects of cirrhosis, aged 20, 30, 32, 42, 44, 54; 5 suffered from malignant disease, their ages being 30, 44, 48, 51, 59.

Among the females there is no case of gastric ulcer. One girl, aged 17, was the subject of cirrhosis; 2 suffered from malignant disease, aged 56 respectively; and 1 child, aged 10, had cardiac disease. In each case where the cause of hæmorrhage was found to be due to cirrhosis, no ulceration or breach of surface could be discovered in the walls of the stomach.

These cases would seem to endorse fully my opinion *that hæmorrhage from the stomach in early adult female life is usually not a symptom of great gravity.*

They, moreover, bring out the striking fact that during a period of twenty years at one of our largest London hospitals there has been no *recorded* case of fatal hæmorrhage occurring in the person of a young female the subject of gastric ulcer.

These cases also establish an important clinical fact—the frequency of severe hæmorrhage in early life as the result of cirrhosis. This cause is one I believe very frequently overlooked, and may be considered as common among women as among men. Brinton, in dealing with the symptom hæmorrhage, mentions that he has found recorded 57 cases where death resulted from hæmorrhage.* The same search gave 233 cases fatal from perforation. Of the 57 cases in which hæmorrhage proved fatal, 18 only were females, 34 being males. The average age of females was over 40. Habershon gives the number of fatal cases occurring at Guy's Hospital during twenty years; they are 65 in number—37 males and 28 females.

He tabulates the cause of death as follows:—Hæmorrhage: males, 9; females, 4. Perforation: males, 12; females, 14. Various, excluding hæmorrhage and perforation: males, 16; females, 8. The ages of the patients dying from hæmorrhage are: males, 28, 43, 53, 53, 58, 60, 60, 63, 63; females, 20, 50, 50, 55.

Among the 155 cases of severe hæmatemesis collected for me by Dr. Goodall, the large number 66 were considered due to gastric ulcer. Of these cases 29 were patients under 30 years of age, 2 only being males. Between 30 and 40 years of age are 21 cases, 11 being of the male sex.

Among those fatal cases where the hæmorrhage proceeded from ulceration extending into a vessel, there is but 1 patient under 30 years of age, a male, aged 23, also the subject of phthisis and pyo-pneumothorax. In cases proving fatal in early adult life, the hæmorrhage was invariably due to cirrhosis.

During the twenty years 1870–90, there were 16 patients admitted into Guy's Hospital suffering from peritonitis, the result of a perforating gastric ulcer. Eight of these patients were males

* 'Ulcer of Stomach,' p. 47.

and 8 were females; the ages of the females being 15, 14, 21, 22, 28, 34, 40, 67; the males, 19, 28, 40, 43, 45, 50, 51, 62.

During the twenty years tabulated by Habershon, I find there were 18 similar cases admitted into the hospital: 11 were males and 7 females. Ages of males: 28, 31, 34, 38, 42, 59, 62; females, 21, 22, 22, 27, 37, 40, 63.

In a paper published in vol. xxxi of the 'Transactions of the Pathological Society,' Dr. Norman Moore brings forward the fatal cases of gastric ulcer occurring at St. Bartholomew's Hospital during thirteen years, 1867-79. There are 14, 11 being males and 3 females. Death in 2 of these cases was caused by hæmorrhage, both the patients being of the male sex, one aged 19, the other 57. The ages of the other cases were as follow: males, 19, 36, 40, 41, 46, 47, 57; females, 46, 47, 54.

In vol. xxi of the 'Transactions of the Pathological Society,' are two cases recorded by the late Dr. Murchison. They have been frequently referred to in asserting the fact that severe hæmorrhage may occur from the stomach with but very slight ulceration.

The first case is that of a woman aged 50, a confirmed and heavy drinker. She suffered from very severe hæmatemesis, so severe that, according to the report, upon admission into the hospital her clothes were found to be soaked with blood. After admission she had another attack of hæmorrhage. The hæmatemesis occurred at intervals up to the death of the patient. At the *post-mortem* examination a very small hæmorrhagic erosion was found. In the centre of this minute ulcer there was an eminence leading by a pore-like opening into one of the gastric vessels.

The second case is that of a young man aged 28, a soldier, with syphilitic and intemperate history. He was seized with repeated attacks of hæmatemesis. After death the hæmorrhage was found to have arisen from a small superficial erosion about the tenth of an inch across. Into the centre of this ulcer, a branch of the gastric artery opened by a pore-like perforation.

Fagge alludes to these two cases in pointing out the extreme difficulty in distinguishing between hæmatemesis from simple congestion, and the hæmorrhage arising from ulceration; he sums up with the opinion that he considers a differential diagnosis impossible.

I believe that such cases will bear a different interpretation. I

think they point especially to the extreme danger connected with an ulceration which *has* extended into a vessel; however small the perforation, a recurrence of the bleeding is prone to take place.

A severe case of recurrent hæmatemesis was admitted under my care into the North-West London Hospital during August, 1891. A young woman had suffered for some months from the ordinary symptoms of gastric ulcer, but the dyspeptic symptoms were not accompanied by hæmorrhage. On the day of admission she had had several attacks of hæmatemesis; she was seized with hæmorrhage in the streets and was admitted in a dying state. A *post-mortem* examination revealed the presence of a large ulcerated surface. In this case there was not marked anæmia, and the gastric symptoms previous to hæmorrhage were insignificant.

It certainly appears to me strange that, having regard to the large number of cases of severe hæmatemesis occurring in young female life, so few should be found proving fatal. On the other hand, the *post-mortem* records of every hospital in the kingdom will furnish abundant proof of the fatality attending the simple gastric ulcer through perforation and peritonitis. If the hæmorrhage of young female life be invariably or even frequently due to ulceration, we should, I think, find it more often followed by fatal results.

The very fact that the ulcer had extended into a vessel shows that the process is an active one. But the clinical history of many of these cases points to a different conclusion. The hæmorrhage is often followed by an amelioration of all symptoms referable to the stomach lesion. I have been frequently assured that subsequent to the hæmorrhage all pain and discomfort had ceased. This could hardly be the case if active ulceration were still in progress. Many cases of copious hæmatemesis have been explained by the assumption that the ulceration was superficial. But it is inconceivable, to me, that any such superficial loss of tissue could cause such a sudden and severe discharge of blood as is undoubtedly present in numerous instances. It is in cardiac disease that such superficial erosions are commonly met with, but it is comparatively rare to find them accompanied with copious hæmorrhage. It may be urged that the fact of the patients being placed under treatment is sufficient to account for the improvement, but against this view I have often found patients applying for hospital advice some days after the hæmorrhage. And I have

ascertained that the relief had followed immediately upon the attack of hæmatemesis.

In bringing before your notice the cases collected for me from Guy's Hospital, I stated that each case was an undoubted one and verified in the hospital, all cases in which the patient's own statement that hæmorrhage had occurred previous to seeking advice being excluded. This precaution, necessary when the mere case books of a hospital are consulted, will naturally exclude the large majority of patients with which my communication deals. In numerous instances the hæmorrhage has occurred before the patient's admission, and the only cause for their application for medical advice is the fact of this initial hæmorrhage.

My own personal experience deals with about 40 cases of hæmatemesis occurring among women. Of these cases I have notes of 33 patients. I cannot affirm that in each case the hæmorrhage occurred while the patient was actually under observation, but in each case I satisfied myself that a decided and copious hæmatemesis did take place. It is a difficult matter following the history of hospital patients, but as far as I know all these cases remained well after discharge from hospital. One patient, a lady, aged 30, came under my notice in private work during January, 1880. Weight and discomfort attending digestion had been complained of for some weeks; there had been no acute pain or vomiting; the bowels had been obstinately confined. During dinner this patient was seized with sickness. The fluid vomited was blood, which was seen by me, and estimated at about 10 ounces. The hæmatemesis was followed by marked relief to the gastric symptoms. There was no recurrence, and up to the present date there has not been any return of indigestion. The patient was treated with saline aperients.

It cannot be disputed that the mucous membrane of the stomach will bleed readily and profusely without there being any breach of surface. Among the cases of hæmorrhage admitted into Guy's Hospital were 44 considered as being due to cirrhosis. Fifteen of these patients died, and in no case was there any abrasion or ulcer to account for the hæmatemesis. It must not be forgotten that these examinations were made by some of the leading pathologists of the day. Such hæmorrhage may occur in early life. One case was that of a young girl, aged 16, and two youths, aged respectively 17 and 18, succumbed to this form of hæmorrhage.

There are many cases bearing on this point published in the various journals and reports. A very interesting and instructive one is to be found in vol. ix of the 'Transactions of the Medical Society.' It is brought before the Society by Dr. Stephen Mackenzie. The patient, a woman, aged 39, had suffered from repeated attacks of hæmatemesis, with gastric pain and vomiting. She had been in hospital on three separate occasions, and was considered the subject of gastric ulcer. In spite of treatment the vomiting, pain, and hæmatemesis continued. This attack lasted ten weeks, when death terminated the case. At the necropsy no obvious sign of disease could be detected.

A very similar case occurred in my own practice:—

M. G—, a female, aged 43, was admitted into the West London Hospital on January 4th, 1892. The patient stated that a few days before admission she was suddenly seized with hæmatemesis, a large amount of blood being ejected. The attack was followed by extreme irritability of the stomach, the matter vomited being coffee-ground in character. This coffee-ground vomit was noted while the patient was under observation, and occurred on several occasions. The epigastric region was very sensitive to pressure. At first sight all the symptoms and the physical condition of the patient pointed to a gastric ulcer as being the cause of the illness. But when the past history of the case was inquired into, we ascertained that during the preceding four or five years there had been many similar attacks. During the previous year she had suffered from three. These attacks seemed invariably to have followed a condition of extreme constipation. The patient was suffering much from this cause at the time of her admission, and a pultaceous mass could be easily felt in the abdomen. The treatment was directed solely against this state of bowel. The patient quickly recovered, losing all pain, and digested solid food without difficulty. She was discharged on January 30th, being twenty-six days under observation.

Some years ago a lady brought me her daughter for advice. The patient was suffering from anæmia; the blood change had come on rapidly, and was of an intense character. Before leaving my room I was asked, "Is this the form of anæmia in which a blood vessel so often ruptures in the stomach?" I was much struck by the question. I ascertained that in the part of the country, North Devon, from which my patient came, anæmia was a very common form of illness, and was frequently accompanied by hæmorrhage. I wrote to Dr. Drummond, of Dolton, a village near the neighbourhood referred to, and he thoroughly endorsed the statement. He informed me that anæmia among young girls was very common in the district—in fact, he generally had cases under his care; that this anæmic condition was often followed by

hæmatemesis, in many cases the hæmorrhage being profuse. For some time he had regarded this hæmorrhage as being apart from ulceration, and as being but a part of the anæmia; that this opinion had been forced upon him by the rapid recovery these patients made when treated with iron and sulphate of magnesia, and by the fact that the hæmorrhage did not prove fatal. He had had the opportunity of making only one *post-mortem* in a case where, previous to death, the patient had suffered from severe hæmorrhage, and in this case the stomach wall was found intact.

An extremely valuable and interesting case bearing on this question has been kindly furnished to me by Dr. Barton, of Cheniston Gardens. On September 26th, 1890, a young girl suffering from anæmia, aged about 21, a nursery maid, applied at his house for advice at 10 A.M. She stated that while walking in the park she was seized with vomiting and threw up about a tea-cupful of blood. At 1 P.M. Dr. Barton was summoned to the house where the patient was in service. He found she had had a very severe attack of hæmatemesis, an immense quantity of blood being ejected. The patient was transferred to University College Hospital in an ambulance. While there a recurrence of hæmorrhage took place. A few days later the patient was seized with "mumps," and died from hyperpyrexia. A most careful and exhaustive examination was made under the supervision of Dr. Ringer, but no source of bleeding could be discovered in œsophagus, stomach, bowel, or lung.

Apart from the main symptom, hæmatemesis, the chief clinical features of the cases to which my communication refers are much the same—indigestion, vomiting, abdominal pain, referred to the region of the stomach, usually obstinate constipation, and more or less anæmia, with scanty catamenia. In brief, such an array of symptoms, when supplemented with an attack of hæmatemesis, is regarded as being pathognomonic of gastric ulcer.

From careful clinical observation, is such a conclusion invariably warranted by the facts? In some cases at least I feel the argument is somewhat strained. Briefly, it is much as follows: Young women are well known to suffer fatal peritonitis due to the perforation of a gastric ulcer. Young women are frequently the subjects of severe hæmatemesis, often accompanied by more or less gastric discomfort. Severe hæmorrhage is found occurring during the course of gastric ulcer. *Ergo*, the hæmorrhage of

young women is due to such ulceration. This argument cannot be supported by morbid anatomy.

Profuse hæmatemesis may occur without any marked disturbance of general health. Murchison remarks:—"Hæmatemesis from cirrhosis often occurs, the patient being in fair or good general health." The most profuse case of hæmatemesis I have seen was in the case of a patient I saw in consultation in 1884. The patient, a gentleman, aged 74, previous to the attack had complained of a weight at the stomach and an indefinite sense of indigestion. He had had no true pain and no vomiting. Of full and luxurious habit, he had taken but little exercise. Suffering from joint pain, he had consulted a physician for gout, and had been advised to walk daily from 4 to 5 miles. He followed the advice for a week or so; his ankles began to swell, and the stomach trouble commenced; a violent attack of hæmatemesis being the final symptom. When I saw the patient he was completely blanched by loss of blood. A perfect recovery took place, there being no return of hæmorrhage or gastric trouble up to death, which took place eight years later.

Hæmatemesis in young women is a symptom so constantly associated with profound blood changes—anæmia and chlorosis—that there would appear to be good grounds for regarding the association of symptoms as more than a coincidence. It is, moreover, a well-known clinical fact that these blood changes almost invariably precede the discharge of blood from the stomach.

There are observers who consider the anæmia to be the result of mal-nutrition, due to the supposed stomach lesion. Brinton stoutly maintains this view, and in discussing the relation between anæmic conditions and gastric ulcer states as his opinion that the blood condition is absolutely caused by the state of the stomach. Mark his words: "The anæmia produced by the hæmorrhage is generally associated with a cachexia which seems to be essentially independent of it; being chiefly the result of the inanition necessarily implied by frequent vomiting of the food, or by the large destruction of the gastric mucous membrane and consequent impairment of its function."

It appears to me that Brinton is here confusing the two classes of case. Clinically, the symptoms denoting extensive ulcerative surface are markedly different from those found in anæmic young women. Among this latter class we more generally

find the punched-out ulcer, and how often such an ulcer is unaccompanied by any symptom at all ! Without doubt the constant vomiting seen in many cases of extensive gastric ulceration may speedily bring the patient to the verge of starvation, and here the inanition is directly due to this loss of nourishment. But with many of the patients suffering from hæmatemesis and anæmia there is no such loss of food and no wasting. Those who have had clinical experience of these cases will uphold me, I feel sure, in asserting that the anæmic cachexia or chlorosis differs essentially from that seen accompanying cases of inanition. Many of the young women suffering from hæmatemesis are plump and well-conditioned, but their waxy unhealthy-looking skin denotes blood change of a far more complicated order than that seen in simple starvation. If we examine closely and particularly the symptoms existing in undoubted cases of ulceration, and compare these symptoms with those met among anæmic girls, we shall, I think, in a large number find a very striking difference. The symptoms to be considered are pain, vomiting, and the varied forms of dyspeptic trouble. Let me briefly refer to one of the last cases of hæmatemesis under my own care.

M. P—, a young woman, aged 19, admitted into the West London Hospital on November 4th, 1891. Three days before admission she had an attack of hæmatemesis, and was stated to have lost a very large amount of blood. The marked anæmia or bloodlessness of the patient warranted the truth of the statement. The hæmorrhage recurred twice, the three independent attacks occurring within forty-eight hours. She stated that she had been out of health for some weeks, and noticed her colour had changed. She was well nourished, but of extreme waxy hue. During all this period she suffered more or less from indigestion, which was expressed by discomfort, rather than pain, after food. She had vomited after food on two occasions only. Her bowels had been constipated, and the catamenia scanty.

After the attack of hæmorrhage the gastric pain entirely left the patient, she had no sensation of weight at stomach, and had no sickness. The bowels were very obstinately confined. Placed on light food for a week, with a mixture containing sulphate of magnesia and infusion of gentian, the patient, beyond being weak, expressed herself as feeling perfectly well. On the eighth day she was taking ordinary food ; on November 16th was up and about the ward ; a week later discharged as well. May not such an attack of hæmorrhage be analogous to that seen in recurrent epistaxis ?

In those cases where I have verified the actual presence of gastric ulcer by *post-mortem* examination, and where, previous to death, the patient has been any length of time under observation,

I have been struck with the marked difference of their gastric symptoms as compared with such cases as the above. With regard to the pain, I never remember in any single instance seeing such an acute pain—agony, it may justly be called—as found with patients the subject of gastric ulcer.

From my experience the gastric trouble of these young women rarely amounts to more than discomfort—a weight, an uncomfortable sensation, coming on gradually after food, doubtless often accompanied by vomiting and relief; but the vomit differs materially from that seen in true ulceration. I believe it is seldom large, or consisting of that intensely acid watery fluid so frequently met with in ulceration. In several cases I have found the sickness which has been complained of to be nothing more than eructations of gas, accompanied by some of the contents of the stomach. How frequently such a symptom is complained of by anæmic girls who do not suffer from the crucial symptom hæmatemesis!

Subjects of true ulcer often complain of water brash of intense acidity; they are apt to suffer from recurrent attacks of stomach irritation, at times so extreme that no food is borne in the viscus for days. My own personal experience does not lead me to think such symptoms are usually met with in that form of dyspepsia accompanying anæmia, and so often followed by hæmatemesis.

Perforating gastric ulcer is not infrequent in the male sex.

From the cases brought before you the liability of both sexes is about equal. Why should not hæmorrhage be as usual in young adult male life as it is in a corresponding period of female life? Yet profuse hæmatemesis, except as the result of cirrhosis, is practically extremely rare in young men. The dynamics of their circulation are profoundly different.

It has not been my intention in this communication to bring before the Society a detailed description of those cases of hæmatemesis which have been under my own care. The clinical features of such cases are common property with us all. In no sense am I attempting to describe a new disease, rather I am begging your forbearance in permitting me to examine more closely the etiology of a very old one.

If it can be shown that in even a small proportion of cases of severe hæmatemesis occurring in early adult female life, the hæmorrhage need not necessarily be due to ulceration, but may be dependent upon more complex functional changes, apparently

underlying the anæmic state, the treatment of such cases will undoubtedly be very different to that called for in gastric ulceration.

The feeding of these patients must be based upon an entirely different system to that demanded by gastric ulceration; and in a large proportion of cases we shall find the troubled digestion, the various forms of dyspepsia, pass rapidly away under a course of saline aperients combined with preparations of iron.

I have noted that in many of these cases there has been, previous to the hæmorrhage, an extreme state of constipation. With such patients I find a mixture containing sulphate of magnesia and perchloride of iron is usually efficacious, the dose of magnesia being regulated by the exigencies of the case.

Gentlemen, in bringing before you in a but very brief and imperfect manner my views on this "*Hæmatemesis Puellaris*," I would ask your indulgence. The whole subject is an abstruse one, and beset with difficulties and conflicting equivocal symptoms. We meet with cases in which every classical symptom of gastric ulcer is present, and yet on the *post-mortem* table the stomach wall is found to be intact. We meet with cases where there is no gastric symptom worthy of name, and yet an ulcer has caused the death of the patient. Again, we meet with cases where, in the opinion of the best observers, there is no symptom warranting the diagnosis of ulcer, but where an ordinary dyspeptic condition is eventually found to have been caused by extensive ulcerative process.

Personally, I cannot help feeling that much of the ambiguity attending the diagnosis of gastric ulcer is largely owing to the undue weight attached to the symptom *hæmorrhage*. Many a case of gastric ulcer runs its full course with no trace of bleeding from the stomach, and I believe the converse to be equally true, and that many cases of copious hæmatemesis occur without there being a breach of stomach wall. We know this to be true of cirrhosis, and I trust I have brought before you this evening sufficient evidence that the hæmatemesis of early adult female life need not necessarily be due to ulceration of stomach, but that such hæmorrhage is possibly the result of venous congestion, or stasis in the vessels of the œsophagus or stomach mucous membrane—a state of sluggish circulation depending upon complex changes of blood, and expressed generally by what we know as anæmia. I venture

to think that, hypothesis though it be, it is an hypothesis based upon more than mere conjecture; and, moreover, it will be found to be a working one and one we can make use of at the bed side—one which, if acted upon in a fair proportion of cases, will contribute more help to our patients than by subjecting them to that rigorous treatment essentially called for in gastric ulceration.

Dr. HABERSHON thought that the Society was indebted to Dr. Donald Hood for bringing before them this interesting class of cases of hæmatemesis in young females. He was obliged, however, to join issue with the author on one or two points. In the first place, the weak point of a hypothesis which excluded gastric ulcer as the common cause of the hæmorrhage in such cases was that, on the one hand, it was impossible in most instances to follow up the cases or to confirm the diagnosis *post mortem*, as they usually recovered; and, on the other hand, the opinions of well-known authorities must be weighed against it. With regard to his father, whose name had been mentioned, Dr. Habershon was able to supplement the statistics given by Dr. Hood by a word or two with reference to the cases found in his private note-books, the statistics from which had just been published by him in the 'St. Bartholomew's Hospital Reports.' He would say, with regard to Dr. Hood's statement, that hæmatemesis accompanying this group of symptoms was far more common in young females, that there was undoubtedly a large class of cases also occurring in young men, for no less than forty cases of hæmatemesis, mentioned in his father's note-book, occurred between the ages of 20 and 40. These were presumed to be cases of gastric ulcer. Doubtless the cases were far more frequently to be met with in young women. With regard to diagnosis, he must mention that he had looked carefully through the case books in order to discover whether a class of cases of hæmatemesis were believed by his father to have occurred of (in his judgment) obscure diagnosis. He was unable to find more than one or two of these, and they were of a character well illustrated in his work on 'Diseases of the Abdomen' of hæmatemesis occurring in young hysterical, and, sometimes, anæmic women with amenorrhœa. The hæmorrhage was said to occur at the time of the menstrual period (the so-called vicarious menstruation). The weak point, however, of the cases was that the hæmorrhage never occurred while the patient was under observation, and was always a matter of history only. All the other cases (excluding such as were manifestly due to hepatic disease or portal congestion) were diagnosed under the category of simple ulcer of the stomach or of superficial ulceration. The difficulty of diagnosis was, doubtless, increased by the fact that hæmatemesis from gastric ulcer was sometimes accompanied by the typical symptoms (pain, vomiting, &c.), while in the other cases hæmorrhage might occur in the first instance without any such preceding symptoms. Dr. Habershon was sorry that he was unable, without further evidence, to agree that the majority of such instances as the author had mentioned were to be explained by causes other than one of the forms of gastric ulceration.

Dr. SIDNEY PHILLIPS thought the two main points to which Dr. Hood had drawn attention were the existence of ulcers of stomach without hæmatemesis and the occurrence of hæmatemesis without ulceration. With reference to the latter, Albert, long ago, had described cases in which violent pulsation of the abdominal aorta was associated with hæmorrhages

from the alimentary canal. He (Dr. Phillips) had recorded similar cases in a paper published in the 'Brit. Med. Journal,' 1887; possibly mere vaso-motor disturbance might give rise to the hæmorrhages.

The PRESIDENT alluded to cases which had proved fatal, and in which no lesion had been discovered *post mortem*. He referred to a specimen in the Middlesex Hospital museum in which a minute aneurism had ruptured, and suggested this as a possible cause. He noticed that patients usually got well rapidly after the occurrence of hæmorrhage, possibly through the clot affording some protection to the site, and if allowed a few days rest by avoiding stomach medication and feeding, enabling the ulcer to heal underneath the blood scab.

In reply, Dr. HOOD stated that at the late hour of the evening he did not intend to offer more than the briefest remarks. The paper stated fully his views on the subject under discussion. He did not expect the members present to agree with all these views, but he did wish to attract their attention to the subject. Time only could show whether the deductions he had drawn from clinical observation were legitimate or not. With regard to the issue raised by Dr. Habershon, that hæmorrhage from the stomach was *not* uncommon among the young of the male sex, he could only say that, as far as his own personal experience went, it was a symptom of extreme rarity. The period of life referred to, when hæmatemesis was usually met with among girls suffering from anæmia, would approximately lie between 17 and 25 years of age. Excluding cirrhosis, hæmatemesis in the male sex at this time of life is not common; and this statement will be found to be strongly supported by the cases brought before the Society in the paper just read.

February 29th, 1892.

THE DIAGNOSTIC AND PROGNOSTIC VALUE OF TUBERCLE BACILLI IN THE SPUTUM.

By FRANK J. WETHERED, M.D.

SINCE the discovery of the tubercle bacillus in 1882, by Koch, much has been written in regard to the value of a search for this organism in the sputum and other fluids of the body as an aid to diagnosis. The subject has only been twice brought before the English medical societies. Dr. Whipple read a paper before this Society in 1883, and Dr. Percy Kidd and Mr. Taylor presented a valuable review of the matter before the Royal Medical and Chirurgical Society in 1888. In that paper the authors proved conclusively the great diagnostic value of the discovery of the tubercle bacillus in the sputum.

I should like to make a few additions to what has already been published on the aid to diagnosis furnished by the tubercle bacillus from my own experience, and also to comment on its prognostic signification.

First, then, a few words as to the method of staining. After trial of several methods, I have always reverted to the Neelsen-Ziehl process, that is, staining in a carbolic acid solution of fuchsine and decolorising in a 25 per cent. solution of sulphuric acid. One of the most important points is the selection of the particle of expectoration to be examined. The sputum coughed up early in the morning before any food has been taken is to be preferred. I have found bacilli very plentiful in such a sample, whilst another taken from the spittoon haphazard during the day has yielded negative results, or bacilli were only found sparsely scattered through the specimen, several of which had to be examined before the organisms were seen.

Any particles of food, especially flakes of milk or small particles of starchy matter, add materially to the difficulty of selecting the most suitable portions. I have received samples of sputum so mixed with food matter that a satisfactory examination was out of the question. It is advisable, therefore, to request the patient to wash out his mouth before using the spittoon.

The specimen should be thrown out into a flat dish, preferably a black one; those used by photographers suit admirably. Search must then be made for the small yellowish points so common in phthisical sputa, or if none of these can be found for opaque streaks and small masses, which are more common; failing these, the most solid looking points should be chosen. The selected particle must then be removed to a clean cover-glass; this is most conveniently accomplished with the aid of a pair of forceps and scissors, the forceps having one blade bent into a short hook. Two steel pens in holders act very well as teasers and lifters. Another cover-glass is now laid on the first, and by means of a gentle pressure, the sputum is spread out into a uniform layer, any excess that escapes from between them being wiped away with a soft cloth. This layer should be made as thin as possible, as a thick film is difficult to decolorise. The glasses must be separated by sliding them apart, not springing them, otherwise the uniformity of the films will be destroyed. The specimens are allowed to dry in the air, or the process may be

hastened by applying moderate heat; if the glasses are held between the fingers there will be no fear of over-heating them. When dry, the films are fixed to the glasses by passing the latter, prepared side uppermost, three times through a flame in order to coagulate the albumin.

The first staining process consists of laying the preparations, prepared side downwards, for two minutes in the heated fuchsin solution. Two minutes is ample time, and no benefit is gained by allowing them to remain longer. If the stain be cold, an hour is required. They are next decolorised by being placed in a 25 per cent. solution of sulphuric acid. A few seconds usually suffice to take the stain out of everything but the bacilli; owing to their capsule the organisms resist the action of the acid for some little period. It is important that this decolorisation be thoroughly done, in order to obtain good and clear specimens, otherwise blotches of red will remain, which not only obscure the field, but may even be misleading, giving the appearance of rods when none are there. The glasses, therefore, must remain in the acid until all red colour has disappeared. Contrary to what is usually stated, it is difficult to wash the dye out of the bacilli; I have allowed the glasses to stay in this strength of acid for half an hour, and yet found the bacilli stained as well as over. The best mode of procedure is, after removing one of the glasses from the fuchsin, to wash it for a few seconds (about eight or ten) in the acid, and then to swill it in water; part of the colour will probably be restored, as may be ascertained by holding it up to the light; it must then be returned to the acid for some seconds and again washed in water, this process being repeated until no more red colour is visible. It is advisable to counterstain the preparations in order to make the contrast of the red stain more marked, and the most satisfactory colour for this purpose is blue, using a 2 per cent. solution of methylene blue, in which the glasses remain for about twenty seconds or half a minute; they are afterwards washed in water, dried by pressing them between leaves of filter paper, and examined in water or glycerine, or, if permanent preparations be required, in Canada balsam.

The specimens, having been duly stained, are examined by the microscope. In order to obtain satisfactory results, and be sure that if bacilli are present, however few they may be, yet they will be seen, it is important that an Abbe's condenser should be used,

and a twelfth oil immersion lens. The rods can be seen with a good quarter, still better with an eighth, but with greater ease and precision with a twelfth.

What conclusions are to be drawn from the presence or absence of tubercle bacilli? I know that there are some medical men who still view with a considerable amount of scepticism the value which is to be attached to those minute particles of vegetable life, and perhaps still more, who consider percussion and auscultation, together with the clinical history, to be more trustworthy guides than the microscope; but this matter I will refer to again.

I do not intend to enter here into a full discussion of the ætiological connection of the bacillus with tuberculosis, or of that mysterious bugbear to medical science, the second ætiological factor, called according to fancy, "favourable pabulum," "deficient resistance," "predisposition," &c., but will take it for granted that, without the bacilli, tuberculosis cannot exist.

Roughly speaking, I think we may say that in the search for tubercle bacilli a positive result, that is to say, their presence, is everything, whilst a negative, their absence, is practically of no value. But this broad statement requires some modification.

If bacilli are present in the sputum, we are sure that there is a tubercular process proceeding somewhere in the respiratory tract; the exact site will probably be ascertained more easily by the stethoscope and laryngoscope than by the microscope, although valuable information may often be gained by the examination of unstained specimens.

It would be obviously of little use to examine the expectoration for bacilli in cases of phthisis far advanced, where the diagnosis is obvious; but the great value of this process lies with those cases where the physical signs are indefinite; when there are a few suspicious signs at one apex or at one base, perhaps only weak breath sounds with occasional crackle, perhaps also, only slight disturbance of temperature, and the patient denies previous cough. Many of such cases take an insidious course until the disease has considerably advanced, and its true nature is not recognised until the prognosis is necessarily worse than if the tubercular character of the affection had been at once recognised. In such cases, tubercle bacilli can often be found in the sputum from the first, and, consequently, the necessary precautions taken.

Although the characteristic sputum of phthisis is well known, there is no variety in which bacilli are not sometimes found. I have detected them in large numbers in perfectly limpid, watery, mucoid expectoration, resembling that which is met with in the early stages of simple bronchial catarrh. In what condition the lung must be in order that the bacilli can appear in the sputum, I will not stay here to discuss; but for the purposes of this present paper I can only say that I have frequently found them when by physical signs and clinical history alone it would have been very hazardous to have ventured upon the diagnosis of tubercle. In some of the cases the course of the disease has corroborated only too truly the correctness of the discovery, the patient dying within a few months of acute phthisis. One case I remember well, a ward-maid in the London Hospital, who exhibited only slight relative dulness in the right supra-spinous fossa, with occasional crackle after cough; the diagnosis would have been doubtful, but that tubercle bacilli were found in the sputum; a *post-mortem* three months afterwards proved the presence of pulmonary tuberculosis.

There are two other classes of cases in which the discovery of tubercle bacilli is of the greatest aid to diagnosis. When a tubercular lesion of the lung is surrounded by emphysema, or when an attack of bronchitis has supervened, the physical signs of phthisis are often so masked as to be scarcely recognisable, and can only be with certainty diagnosed by the use of the microscope.

The second class of cases to which I refer are laryngeal cases, in which the lungs are healthy, but where the differential diagnosis between tubercle and syphilis cannot be substantiated by the laryngoscope or clinical history. If tubercle bacilli can then be found in the expectoration, the doubt is completely cleared up.

Some observers (Zahn, Ziel, and Leyden) have stated that they have found tubercle bacilli in the sputa of patients not suffering from tubercular disease, especially when these patients have been in the same wards as consumptive patients, arguing, therefore, that the bacilli have merely entered the air passages and not found a suitable nidus. These observations were made in the early days of staining for the bacilli, when the methods were not so exact as they are now; whether these observers still hold these views, I do not know, but certainly they have not been verified by others. Dr. Kidd and Mr. Taylor state that in their very large experience

they have never met with such cases, and, having examined some hundreds of samples myself, I can add my testimony to theirs.

Turning now to a negative result in the examination of sputum for bacilli, we have not nearly such a satisfactory case. From the examination of one sample only, if no bacilli can be found, absolutely no value is to be placed on the result, for the absence of tubercle bacilli does not necessarily imply that the case is not a tubercular one. If repeated examinations are made, every precaution being taken as regards the collection of the sample and the selection of the most likely particles for staining, and the result is still negative, the presumption certainly becomes stronger that tubercle is not present; but even then the evidence is not conclusive. I once examined the sputum of a patient supposed to be suffering from tubercle, twenty-one times; that is to say, every day for three weeks, and only found tubercle bacilli at the twentieth attempt. *Post-mortem* examination showed that the patient died from miliary tuberculosis of the lungs; and this points to an important fact, that in cases of miliary tuberculosis of the lungs I have again and again found that it is extremely difficult to detect tubercle bacilli in the sputum.

As regards prognosis, when bacilli come "they come not single spies, but in battalions," and little can be learnt from their numbers and distribution in the expectoration. The mere fact of their presence naturally increases the gravity of the case; but beyond this I do not think it is safe to go. The general aspect of the case is a surer guide; improvement of the general condition and physical signs, or the reverse, will guide the physician much better than the absence or diminution of the bacilli in any one sample, or even their continued absence for some little time. When we consider the minutiae of the method of examination, it is not to be surprised at. We take an infinitesimal portion of the expectoration only, and there is undoubtedly a strong element of chance as to the particle so selected. It may happen that in a comparatively early case we happen to pick out a small portion of caseous matter which is almost a pure culture, whilst in a more advanced and more acute case, we merely alight upon an opaque piece of mucus, in which the organisms are very few, and these would be obviously false grounds upon which to form an opinion.

Some may say that frequent examination should obviate this, but, according to my experience, I have never been able to trace

any connection between the number of bacilli and the severity or stage of the disease. I have found the rods present in very large numbers when the physical signs were very slight, and when, thanks to the early recognition of the tubercular nature of the complaint, the patient has done extremely well; whilst in other cases, although the signs were distinctly marked and the case evidently progressing rapidly, and although several examinations of the sputa were made, yet bacilli were always few.

The range of temperature also made no difference in the number of the rods.

I am aware that it is usually stated, by Dr. Heron, among others, that if, on frequent examination, bacilli are found to be present in large numbers in the sputum of any one patient, it may be concluded that the case is an acute one; this is no doubt often true; but, on the other hand, one meets with cases in which the patient is rapidly improving in which the physical signs are rapidly disappearing, and yet the same condition of the expectoration holds good. In short, as long as caseous matter is being expectorated, so long will bacilli be found.

No importance can be attached to the grouping of the bacilli, but there is one point upon which I should like to ask the opinion of Fellows of the Society who have had experience in this matter, namely, whether any conclusions can be drawn from the peculiar broken appearance of the rods sometimes observed. These fragments are usually collected together into groups; but if the individual members are closely examined they will be seen to be about a half or a third of the usual length. This appearance, as far as I can ascertain, was first called attention to when tuberculine was introduced, and was thought to be due to the action of that fluid, but this has since been shown not to be correct, as it is sometimes observed in cases not so treated.

From the cases I have seen, I am inclined to think that it is a favourable sign, indicating that the pabulum upon which the bacilli are feeding is not very suitable for their growth. My cases, however, of this kind have been few, and I would not hazard a definite opinion. A week or two ago, I had a sample sent me in which I found this condition, and afterwards heard that it was from an old gentleman aged 75, who had only been ill a few weeks.

The presence of the so-called "spores" has been considered

by some to be an evidence of activity ; but here again I have not been able to convince myself that such is the case.

Hunter Mackenzie has stated that bacilli are particularly numerous when there is tubercular ulceration of the larynx. Acting on this I have sometimes selected such cases for the purpose of demonstrating the method of staining, expecting to secure especially good specimens ; the results have always been disappointing from the point of view for which they were required, and Dr. Kidd and Mr. Taylor also found that in well-marked instances of this type bacilli were decidedly scanty.

Considerable aid in the prognosis, however, may I think be gathered from the examination of unstained specimens of sputum and observation of other ingredients, notably leashes of elastic tissue.

Dr. Troupe, of Edinburgh, goes so far as to say that elastic tissue is often the precursor of the bacilli, but with this statement I cannot agree. I have frequently found bacilli when the most careful search has failed to discover elastic fibres ; and, on the other hand, when elastic tissue is present in cases by other means shown to be undoubtedly tubercular, I have never failed to find the organisms.

My usual mode of examining for these "curled fibres" has been to select several of the opaque particles and place them at once under a cover-glass ; much information can then be derived from the fibres. Dr. Fenwick's method is perhaps more sure, if they are present in very small numbers, but I think to a certain extent destroys their characteristics.

I have often been able to corroborate the valuable observations made several years ago by Sir Andrew Clark. He showed that three distinct forms of elastic tissue can be made out. If the case be very acute and a considerable amount of destruction of the lung tissue going on, complete casts of the pulmonary alveoli make their appearance, often in such large masses as to more than fill two or three fields of the microscope. The fibres are then very elastic, and in every point exhibit their characteristics, especially the interlacing and dichotomous branching. If the case be a more chronic one, the fibres appear more discrete, often occurring singly but with "tailed" extremities ; although presenting their clear double contour, they have lost their elasticity, and care must be taken not to confuse them with chance ingredients such as the

mycelia of fungi, threads of cotton, &c. The third form of elastic fibre denotes a very chronic state of disease; small fragments are then seen more or less incrustated with lime salts, which dissolve on the addition of acid, leaving the degenerated fibres. A fourth variety is also met with, namely, the straight bundles derived from the large bronchi and larynx.

It must be borne in mind that these elastic fibres denote mural disintegration only, and are found in other than tubercular cases, with the exception, as far as I know, of the third form just described.

Before concluding, I should like to refer to two other bodies which I have occasionally, though very rarely, found in tubercular sputa, namely, Curschmann's spirals and Charcot-Leyden crystals. These bodies were formerly considered to be peculiar to asthma, but we have plenty of evidence to show that such is not the case, and those cases of consumption in which I found them were not complicated with asthmatic paroxysms. The spirals seem to denote a catarrh of the finest bronchioles, whilst the Charcot-Leyden crystals are dependent upon decomposition of albuminous substances, their chemical properties showing that they are compounds of the fatty acids.

I trust that the Fellows of this Society will not think that I have brought forward a subject which is hackneyed and not worthy to be discussed, but from conversations I have had with other medical men, there does not yet seem to be any uniformity of opinion as regards the diagnostic and prognostic value of the presence of the tubercle bacillus in sputum, and I therefore ventured to bring the subject once more before the notice of the Society, so that those who have had experience in the examination of expectoration might be led to give the results of their investigations and the conclusions to which they have arrived.

Dr. HADLEY urged the importance of using absolutely clean slides, having found bacilli perfectly stained in slides washed for some days in spirit and potash. The organisms were discoverable in preparations which had been steeped for twenty-four hours in 25 per cent. sulphuric acid. He thought that when bacilli were found, specimens of elastic tissue could always be obtained. By the new methods of staining, however, the bacilli were the more easy of discovery. He agreed as to the difficulty of finding bacilli in miliary tubercle, even when squeezing out the actual miliary granules.

The VICE-PRESIDENT in the Chair (Dr. Stephen Mackenzie) asked

Dr. Wethered whether he had discovered tubercle bacilli in cases of fibroid phthisis.

Dr. WETHERED, in reply, said with reference to the President's question as to fibroid phthisis that he had had very little experience in such cases. In the specimens of sputa which he had examined from cases of supposed fibroid phthisis, he had been unable to find tubercle bacilli. Their absence might possibly be accounted for by the caseous material being encapsuled in firm fibrous tissue. He fully agreed with the remarks which had been made about clean glasses. It was extremely important that the cover glasses should be thoroughly cleansed, but there was less risk of using them again, than in employing glass slides twice over, as would be necessary in Mr. Hadley's process. The difficulty of finding bacilli was certainly increased if the quantity of sputum was very large; hence the importance of selecting the small quantity coughed up early in the morning before any food had been taken. He was glad that his experience of the difficulty of finding the bacilli in cases of miliary tuberculosis of the lungs had been so fully corroborated by the experiences of others.

ON THE INFLUENCE OF NASAL STENOSIS ON THE GENERAL HEALTH.

By W. SPENCER WATSON, F.R.C.S., M.B. Lond.

THERE is good evidence that many forms of disease, not obviously of nasal origin, have been cured, relieved, or prevented by treatment successfully directed against nasal stenosis. This I take as my text; and though I may not be able to prove the statement categorically, I trust that I shall be able to show that it is well founded and sufficiently accurate to form a trustworthy guide in treatment.

It may be well at the outset to give a brief epitome of what is known as to the functions of the nose as a part of the respiratory apparatus.

1. The inspired air is warmed to the temperature of the blood or within 1° or 2° F. of that temperature.

2. The inspired air is moistened by watery vapour exhaled from the nasal mucous membrane.

3. The inspired air is filtered, and to a great extent freed from foreign particles and micro-organisms. Some of these become adherent to the vibrissæ and some to the mucous surface, and in time are extruded with the mucus; the more irritating vapours or micro-organisms exciting a free flow of fluid mucus, which in extreme cases is expelled by sneezing or reflex cough.

4. The temperature of the blood is lowered by the evaporation from the pituitary membrane.

5. The expired air contains some carbonic acid (an appreciable trace), due to the interchange of gases in the nose independently of those due to oxidation in the lungs. There is also probably an evolution of other animal products from the same source.

These being the chief functions of the nose as a part of the respiratory tracts, what modifications or interruptions are brought about by stenosis? This latter condition may be temporary or permanent, partial or complete, and the effects must vary according to each variety or phase of the obstruction.

a. Temporary stenosis occurs in acute rhinitis of any kind, in simple chronic rhinitis, and in congestive irritation of the pituitary membrane from any cause, *e.g.*, that of the presence of a foreign body, of rhinoliths, or of sequestra of bone. The turbinated bodies are the parts more especially liable to swelling under these circumstances by reason of the structure of their submucous erectile tissue. The stenosis due to turgescence of this part of the membrane is distinguished from that due to hypertrophy, partly by the clinical history of the case, but more precisely by the appearance and behaviour of the swollen part, which dimples easily when pressed upon by a probe, and also by the fact that it is almost always speedily reduced in extent and bulk by the application of cocaine.

b. Permanent stenosis is chiefly the result of chronic rhinitis with hypertrophy, and often of intranasal growths and polypi. Distortions and malformations of the septum, naso-pharyngeal growths, adenoid vegetations of the naso-pharynx, enlargement of the tonsils, thickening of the soft palate, congenital and traumatic deformities, are the more common causes of permanent stenosis.

c. When the obstruction is only *partial*, as is often the case, the breath channel being narrowed only, but not occluded, there is the obvious difficulty of deciding in a given case whether the supposed remote effects such as headache, migraine, asthma, cough, laryngeal spasms, dyspnoea and secondary changes in the larynx, trachea, and bronchi may not be due to irritation rather than to obstruction. A slight amount of swelling of the turbinates brings them or some point of their surface in contact with the septum, and as this part is especially sensitive and rendered still more so by inflammatory changes, the local irritation thus induced may

account for reflex irritation of the deeper parts of the respiratory tract. In some cases it is possible to put this to the test by touching with a probe the suspected area, and if, when this is done, the reflex action is induced, we have some evidence that irritation is more prominently at work than stenosis. The use of cocaine locally applied will also be an aid to diagnosis in the same direction. In the condition of hypertrophy there is no shrinking of the swollen parts as in the case of mere turgescence, and hence any marked relief of the symptoms following the use of cocaine is an indication that there is an area or point of excessive sensitiveness, the irritation of which produces reflex action. When this is the result of these tentative measures, much benefit is often derived by reducing the bulk of the hypertrophic membrane or growth. The application of the electric cautery or of a corrosive acid to the part will be generally sufficient, but if the obstructing part is very far back and involves (as it often does) the posterior extremity of the inferior turbinate, the cold-wire snare or my own ring-knife will be the preferable methods for reducing its bulk.

I here exhibit a drawing from a typical specimen of chronic hypertrophy of the inferior turbinate occurring in a young gentleman, whose life was rendered very uncomfortable by its presence (see Fig. 6, page 311). He was sometimes seized with fits of choking during his meals, and was always in terror from the feeling of obstruction. His mental condition was so depressed that his friends became extremely anxious about him. The operation by the cold-wire snare was followed by complete relief. In this case there was a difficulty in deciding as to how far stenosis alone or stenosis with hyper-sensitiveness of the parts afforded the best explanation of the symptoms. There was no obstruction of the other nostril except during the attacks of acute catarrh, which were very frequent, but the nostril (right) from which the growth was removed was completely blocked by it.

This case was one of chronic hypertrophic rhinitis. In this form of disease not only is the epithelial layer thickened, but the cavernous erectile tissue covering the turbinated bones, and sometimes the submucous covering of the septum, become permanently distended and lose the power of recovering their normal bulk. The glands occupying the submucous layer are also hypertrophied, and the surface of the membrane becomes corrugated and raised into folds or villous prominences, the posterior thirds of the

middle and inferior turbinated bodies being most frequently affected in this way. The anterior extremity of the inferior turbinate and occasionally the anterior part of the middle turbinate, when hypertrophied, are generally uniformly swollen, and when pressed by the probe do not recede. Sometimes the anterior parts have also wart-like outgrowths on their surface, but these are not frequent and occupy only one or two spots or surfaces, differing from those on the hinder parts, where they are uniformly distributed over a considerable surface.

Symptoms.—The symptoms are those of stenosis more or less complete. In the advanced cases the breath channel may be completely occluded; in less advanced stages one or both nostrils may admit of a forced snuffling respiration. There is a constant flow of mucus from the anterior nares, and a backward flow into the naso-pharynx, where the viscid secretions form adherent crusts, which are only detached with difficulty and much “hawking” and coughing. We have, in addition, the usual distress of stenosis, the habitual oral breathing, the snoring and distress of breathing at night, the nasal voice, impairment of hearing, of smell, and taste, and asthmatic attacks, with occasional implication of the larynx. The uvula and velum become thickened, and the elongation of the former gives rise to chronic irritative cough. The breath is sometimes offensive from the accumulation of the mucous secretions in the naso-pharynx, and there may be chronic pharyngitis due to the same cause.

The following cases are good illustrations of chronic hypertrophic rhinitis and of its effects on the general health:—

CASE 1.—Mrs. E——, aged 54 years, very tall and stout, and with symptoms of stenosis and prominence of eyeballs. Great mental depression and fainting fits. Much difficulty of breathing at night.

Extensive hypertrophy of both inferior turbinates.

Ether given, and inferior turbinates removed entirely from end to end at two operations.

Great relief. In the course of the next few months caustics applied and subsequently *vulcanite plugs* for reduction of the hypertrophy of the middle turbinates.

Much satisfaction expressed by the patient. The improvement remained for several years, but whenever there were threatenings of a return of stenosis, the *plugs* were resorted to, and with much relief on many occasions.

Within the last few weeks (eight years after the first series of operations) this lady has had a recurrence of the old symptoms due to growths from the outer wall of the right nostril. I removed these under the

influences of cocaine by means of the cold-wire snare, and the result has been perfectly successful, the nostril being now quite free from obstruction. The growth was polypoid with glandular hypertrophy, and arose from the middle turbinated.

It is instructive to remark that though the first operation seemed in this case to be very free, yet it is evident from the sequel that even the total extirpation of the inferior turbinated bone was insufficient, and that it would have been better to have operated still more freely in the first instance.

CASE 2.—A gentleman's gardener, aged 35 years. Symptoms of stenosis for six years. Operations for polypus, but without relief. Fimbriated outgrowth seen from anterior nares on the anterior third of the inferior turbinate. No posterior rhinoscopic view obtainable, but by digital exploration soft growths felt protruding into pharynx from choanæ.

By ring-knife operation (under anæsthetic) the growths depicted in drawings removed at two operations, Figs. 1, 2, 3, and 4. Three years after the patient remained perfectly free from nasal trouble, and immensely improved in health, the commencement of his relief dating from the time of the operations, and having steadily continued ever since.

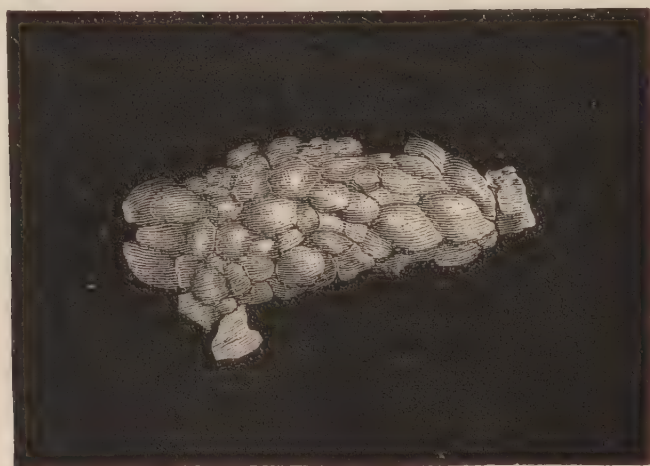


FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

CASE 3.—Mr. H——, aged 50 years. Symptoms of aggravated stenosis for years. Cleft palate. Good rhinoscopic view of growths on hinder extremities of inferior turbinates, complicated with nummular polypi.

Cocaine (20 per cent. solution) applied freely and thoroughly. Numerous polypi removed by cold wire snare. Ring-knife operation on inferior turbinates. Portion of the hinder extremity of the inferior turbinate of the right nostril shown in the drawing, Fig. 5. The size of this growth is somewhat exaggerated in the drawing. It was, however, extraordinarily large. The results of treatment were excellent and lasting. The polypi removed were some of them *nummular* in shape.



FIG. 5.

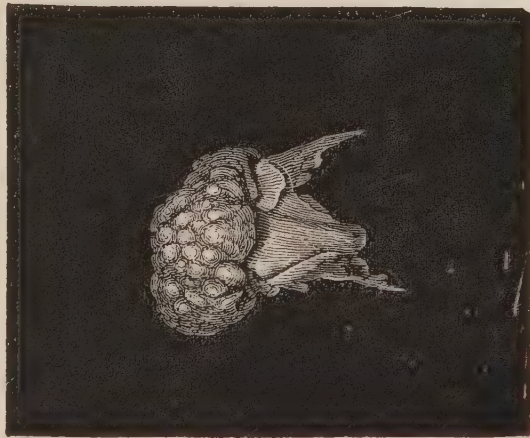


FIG. 6.

In such cases as those related, part of the distress and nervous disturbance is occasioned by frontal headache and a feeling of distension due to retained secretions in the sinuses and ethmoidal cells. The pressure of the intranasal growths obstructs the orifices of the antrum, the frontal and sphenoidal sinuses, the lachrymal sac and nasal duct, and the general hypertrophy of the membrane may even lead to their permanent closure and a large

accumulation of mucus. As a secondary result, sleep is disturbed, and the patients wake up from horrible dreams or nightmare. This deprivation of natural refreshing sleep leads to general nerve disturbance, and migraine and mental depression, so that the stenosis not only impedes free breathing, but at the same time interferes with the functions of the sinuses, and thus gives rise to secondary disturbance of nervous system.

These complications of stenosis make it the more imperative that any operations for its relief should be thorough and effective, so that *free drainage of the sinuses* may be quickly established. It is on this account that I think it very essential to perform radical and complete operations at one sitting under a general anæsthetic, and I suspect that our failures are sometimes due to the frequent repetition of partial and incomplete operations such as are much recommended by some surgeons who employ the electric cautery for the removal of polypi. Each time a small polypus or portion of a polypus is removed, a certain amount of inflammatory swelling is set up, and for a time the stenosis is increased. The patient not being under a general anæsthetic, it is impossible (except in the very rare cases of a single polypus or a very limited hypertrophy) to remove all the growths at one sitting, and few patients will submit to the numerous and repeated operations which become necessary when employing this method. The rule should be—(1) When there is complete obstruction with much constitutional disturbance operate by a single operation under a general anæsthetic. (2) When the obstruction is partial and the symptoms unimportant, the growths being small and easily reached, operate either by means of the snare or electric cautery, using cocaine and employing the frontal mirror as a guide to the instruments. Except in the simplest and most favourable cases, a repetition of the use of the snare or cautery is absolutely necessary.

When both nostrils are occluded and buccal respiration is therefore a necessity, the lungs receive the air cooler and drier, and discharge it with a smaller quantity of carbonic acid and animal excretory vapours than is the case in free nasal respiration. Unless there is a compensatory increased activity in the other excretory organs the blood becomes overheated, and as the lungs are embarrassed in their action, the excretion of carbonic acid and watery vapour is more and more difficult until the blood itself becomes overcharged with these products.

Irritation of the larynx, trachea, and bronchi follows, and in the worst cases bronchitis results.

In the generality of cases the effects of stenosis are not so severe. As to asthma, I have only had three cases in some hundreds of cases of chronic hypertrophic rhinitis. One of my cases of asthma associated with polypi and hypertrophy of the turbinates has been already briefly related in this room. A stoutish married woman, 35 years of age, had suffered from undoubted spasmodic asthma for eighteen years. When I first saw her about two years ago she had numerous polypi and advanced hypertrophy of the turbinates, but these had been previously discovered, and yet for various reasons no operation had been proposed. Here the asthma seemed to be of a confirmed type, and was, moreover, complicated with vesicular emphysema and frequent attacks of bronchitis. Nevertheless, I advised operation, and removed polypi and hypertrophied membrane freely and thoroughly by two operations.

The result exceeded my expectations and those of my colleagues who watched the case. For between five and six months there was not a single recurrence of the asthmatic paroxysms, and the general health improved so much that the woman was able to go to work as a domestic servant, though for many years before she had been incapacitated. For some months she was able to do some very hard work, but in April, seven months after the first series of operations, she had a recurrence of her old ailment. I found some broad based polypi which I have removed by several repeated operations with only partial relief of the asthma. After the last operation on November 5th, she had a severe attack of bronchitis, and great oppression of breathing with regular midnight attacks, and these were controlled only by the use of 8-grain doses of citrate of caffeine. There has been no attack now since December 10th, but she is again suffering from subacute bronchitis and the dyspnoea consequent on the vesicular emphysema. It is quite clear that in this instance of apparently confirmed asthma much benefit resulted from the employment of local treatment. In most cases, however, within my experience, brilliant results are more than can be expected or hoped for. Bosworth, of New York, indeed, and Schmiegelow, of Copenhagen, have had extraordinarily successful cases, the former having cured or relieved seventy-two out of eighty persons operated on for asthma or hay-asthma.

Hay-Asthma.—As for hay-asthma, the disease is obviously one

involving the general system and the respiratory mucous membrane as a whole as well as that of the conjunctiva and lachrymal passages. To consider it as a disease originating in the mucous membrane of the nostril would be to ignore all common experience and the outcome of the most careful inquiries by the most competent observers. The stenosis observed in some of the subjects of hay-fever is due to the effects of chronic hypertrophic rhinitis, which is itself a consequence of the repeated catarrhal attacks so characteristic of the disease. This stenosis is a great aggravation of the other symptoms, and much benefit is often derived from operative treatment, as the suffocative attacks are much mitigated by a restoration of free nasal respiration. The subsequent use of plugs is especially indicated in this form of obstruction, and should be persevered in for many months and even years; this a point not often insisted on in books; I think it very important.

The form of stenosis most interesting to the practitioner is probably that seen in newly-born infants. A poor little child comes into the world with the instinct of getting its living by suction for the first few months of its existence, and with the idea that it will not be asking too much to be allowed to breathe at the same time. But in a few days, or even in a few hours, after birth, "snuffles" of a virulent type sets in, and a cruel destiny declares that the child shall not suck and breathe at the same time—the one line of business may be carried on by itself but not the two together, and as both are essential, the suckling finds himself in an awkward predicament and begins to inquire why his environments are so unsuited to his physical potentialities—that at least seems to be the correct interpretation of the loud and frequent outcries and violent struggles that become too familiar to the mother and friends. The case is one of great urgency and danger. Not only is nutrition impeded in the ordinary act of sucking, but during sleep, the tongue in sucklings being always during sleep in contact with the roof of the mouth, the child is constantly in danger of being suffocated, and wakes up struggling for breath or falls into convulsions of an epileptoid character. Frequent repetitions of these attacks, if unrelieved, speedily put an end to the child's sufferings.

Energetic treatment, however, at the outset by frequent syringing with appropriate astringent and antiseptic solutions, and feeding by the mouth or by a tube passed into the œsophagus through

the nostrils, almost always brings about a speedy change for the better. In addition to these measures, gum-elastic tubes passed through the nostrils and retained there during sleep enable the little sufferer to get tranquil rest. Should, however, the obstruction be only partial, there is still great risk of much mischief to the chest-walls and to the air-cells from the long persistence of dyspnoea even of a mitigated kind. In the course of time the chest-walls become contracted and flattened laterally, emphysema is produced, and the growth is stunted in all parts of the organism. Early and energetic treatment may and often does prevent these disastrous consequences.

The next most interesting form of stenosis is that connected with adenoid vegetations of the naso-pharynx, occurring as they do mostly in early youth and in young adults; they give rise to a set of well marked and peculiar symptoms when the growths are sufficiently large and numerous as to cause serious obstruction in the posterior nares. When slightly developed and early treated, the only symptom noticed is a somewhat dull articulation, as if from a cold in the head, and some amount of stertor during sleep. The general nutrition, however, does not suffer, and very little notice is taken of the defect, which is, according to my experience, very common. In such slight cases, as the body generally develops the pharynx expands, the general nutrition does not suffer, and the symptoms pass off without leaving any traces or inducing any secondary changes in the thorax. In a number of cases, however, in which the obstruction is allowed to go unrelieved for years and becomes aggravated by frequent catarrhal attacks, not only is the articulation dull and indistinct, but the voice becomes habitually nasal; the *n* and *m* sounds are represented by the *d* and *b* or *l* sounds, and the hearing often suffers, the middle ear even in the worst cases being affected by the extension of catarrhal inflammation along the Eustachian tubes, the pharynx and tonsils are involved, and the latter often much enlarged. Snoring becomes constant at night, and may be associated with paroxysms of a convulsive character, the patient waking up suddenly and struggling for breath. There is mucopurulent discharge from the nostrils, and this is sometimes bloodstained, and a similar flow escapes from the mouth during sleep. The breath is offensive from the collection of puriform stringy mucus in the fauces; the breathing is laboured, and the chest often permanently

deformed by a lateral compression and narrowed also in all its dimensions. The growth is stunted; the mental faculties are, or appear to be, dull. The youth is backward in his studies, and takes little interest either in them or in his games. He goes about with mouth agape, and being often somewhat deaf, is credited with being almost imbecile. The alæ of the nose are flattened and dimpled in a characteristic manner, and some of the patients have a very peculiar way of twitching their upper lips and sides of the nostrils. With these obvious symptoms the diagnosis is easy, and is confirmed by rhinoscopic or digital examination of the nasopharynx, where the soft rounded pea-shaped or oval growths are found lining the lateral walls and roof of the pharynx and obstructing the choanæ by overlapping them. When the disease has reached this stage, the only relief obtainable is by operation for removal of the growths, and even at this advanced stage it is often possible to effect a cure of most of the symptoms, though there is much risk in allowing any of these symptoms, especially those affecting the ear, to go on to such a dangerous extent. The majority of cases, however, when treated energetically give most satisfactory results. A lad who had been considered unfit for anything becomes bright, cheerful, and intelligent, and seems quite a different being after the complete restoration of nasal breathing.

When we come to consider *syphilitic disease* in the nostrils, here the stenosis is of secondary importance, so far as the effects on the general health are concerned, with the exception of the syphilitic coryza in sucklings, in whom the obstruction to breathing becomes a matter of grave danger, not only to health but the life of the patient. It must be dealt with early and energetically, not less by local than by constitutional treatment, into the details of which it would be out of place to enter. In the syphilitic ozæna of adults with caries or necrosis, there is the increased danger of the purulent infection due to the retention of foul discharges in the nostril. Fatal meningitis may result from these causes, and the removal of obstructing pieces of carious bone is often a means of saving patients when in this condition.

The stenoses of *malformed and distorted septum* are often overlooked because though, as a rule, there is some obliquity of the nose externally, there may be a considerable twist of the septum without any visible sign externally. There is, however, a ten-

dency (especially when the twist is sigmoid) for the septum to obstruct both nostrils, and to produce all the usual effects of stenosis, laboured breathing, and snoring at night, choking fits on going off to sleep, contraction of the chest walls, and, later on, asthmatic attacks. I have seen several cases of this kind, and it is supposed by Bosworth, Hack, and many others that a form of hay-fever is one of the results of congenital obstruction due to these various malformations. Readjustment of the septum by operation is certainly very beneficial in many cases; whether such operation will cure hay-asthma, however, is, I think, extremely doubtful.

Besides congenital distortions, the cartilage or bone of the septum may be enlarged and thickened, and may cause obstruction of a serious nature, giving rise to considerable discomfort, and affecting the general health in the same way as the other forms of stenosis. Local treatment efficiently applied is always beneficial, and is urgently called for if it is exciting chronic rhinitis, as it often does, or if the respiration is exclusively buccal in character.

Traumatic, cicatricial, and congenital occlusions from other causes all require attention for the same reasons which apply to the condition already alluded to, and cartilaginous, bony, sarcomatous, and malignant tumours of the parts surrounding the nostrils are often more formidable on account of the obstruction caused by them to nasal respiration.

It would be impossible to go into details of all these forms of obstruction. My object in bringing this subject forward is to emphasise the fact that in certain cases, *not in all*, enormous benefit can be conferred on patients by the removal of nasal obstructions, and I think that it will be allowed that, if in a few only there is cure, yet in a large majority considerable relief is obtainable by suitable surgical measures, that it may be laid down as beyond dispute that (1) in young children we may thus prevent deformity of the chest with its attendant evils, marasmus, and even death; (2) that in youth and early adult life we may prevent permanent deformities of the chest, deafness, impairment of speech, and of the mental faculties; and (3) that in adult life we may prevent and even, in some cases, cure asthma, spasmodic cough, bronchitis, emphysema, intellectual hebetude, and melancholia, &c. I have omitted the consideration of many forms of stenosis, such as occur in the course of diphtheritic rhinitis, primary syphilis of the nasal mucous membrane, tubercular rhinitis, intranasal lupus, glanders,

and others, in which the disease is more important from its constitutional than from its local manifestations, because in them the obstruction is generally of secondary interest, and also because local treatment alone will be of little avail. At the same time, it is well to bear in mind that the same principles will apply to them as to the others, and the neglect of local treatment directed against narrowing of the breath passage will nullify much of the good effect otherwise to be obtained by skilfully directed general therapeutics.

In conclusion, I wish to make it clear that while I insist on stenosis being an important factor in many of the remote effects sometimes called reflex neuroses, I by no means wish to exclude the other factors. I think that in most cases of asthma, laryngeal cough, and spasms there is a clear neuropathic element, without which the local obstruction will have no effect; and I also believe that in some cases, such as those of hay-asthma, a hyper-sensitive condition of the respiratory tract (and of the nose as part of that tract) is much more likely to be the starting point of the remote effects than mere obstruction, though there seems good evidence that, even in these cases, obstruction aggravates the condition. All that I contend for is that intranasal obstruction is often an important element in the class of cases referred to. That it is often overlooked, or, if found, despised or made light of, and that it certainly should be sought for and dealt with by local treatment in a very large class of diseases in which, up to quite recently, its influence has been more or less ignored.

Dr. DE HAVILLAND HALL was of opinion that a general anæsthetic was very seldom required in the removal of nasal polypi. If the risk involved in the administration of an anæsthetic were compared with the moderate amount of discomfort attending the removal of polypi with the aid of cocaine, he felt certain that patients would elect the latter method. He also thought that the operation was carried on more satisfactorily if the surgeon could see what he was doing, which is not the case when chloroform is employed. Dr. Hall dissented from the views expressed by the author of the paper on the Treatment of Hay-Fever. He (Dr. Hall) had obtained such excellent results from the employment of the galvanocautery in hay-fever and paroxysmal sneezing, that he could not help regarding a considerable number of these cases as primarily of nasal origin. At the same time he would by no means under-rate the importance of the neurotic element in the case, and he found constitutional treatment of great service. As regards adenoid vegetations, he quite agreed with the author as to the importance of recognising and treating this condition. In the removal of these growths, Dr. Hall insisted on the necessity of the patient being placed under the influence of chloroform, and the nasopharynx being thoroughly cleared out at one sitting. He mentioned that

some authorities were of opinion that the presence of adenoid vegetations predisposed the patients to nocturnal enuresis and hernia.

Mr. W. SPENCER WATSON replied very briefly, stating that he agreed in the main with all the remarks of the speakers, but that he must maintain his opinion that in certain cases of hypertrophic rhinitis it was absolutely necessary to operate with the aid of a general anæsthetic, and that he could not regard hay-asthma as a merely local malady to be treated by local remedies alone.

March 7th, 1892.

THE TREATMENT OF PILES AND ALLIED AFFECTIONS.

By T. LAUDER BRUNTON, M.D., F.R.S.

SOME diseases are important on account of their severity and of the danger to life which they occasion; others are important on account of their frequency and the amount of annoyance they cause to the patient. It is only in rare cases that piles cause any danger whatever to life, but they are so exceedingly common, so very annoying to the patient, so destructive of his comfort, and occasionally of his temper, that they acquire an importance which justifies me, I think, in bringing the subject of their treatment before you to-night. I shall not attempt to deal with the surgical treatment of this disease, nor can I hope to give you anything very new or very striking in regard to the medical treatment. I rather hope to bring together some simple methods of treatment, preventive and curative, and by exciting discussion to the subject to elicit other means, some of which may be known to one and some to another practitioner, but which may not all be in common use together.

I need not enter minutely into the pathology of piles, which is fully treated in works on diseases of the rectum; I may merely remind you that they consist essentially of a dilated or varicose condition of the vessels, arteries, capillaries, and especially the veins of the rectum which are embedded in cellular tissue of a loose and yielding character, and are covered either by the mucous membrane of the rectum, by the skin outside the anus, or partly

by the mucous membrane and partly by the skin, according as they are internal, external, or intermediate, or complicated, as Mr. Allingham terms them. The blood from these veins returns in a two-fold way into the general circulation. Part of it flows through the *anastomoses* of the hæmorrhoidal with the *pudic* veins into the *vena cava*, while another portion passes up through the intestinal and portal veins. The latter portion has, therefore, necessarily to pass through the liver before it can reach the general circulation, and this is a point of great practical importance, because the condition of the liver seriously affects the circulation in the rectum, and an impediment to the free flow of blood through the liver may tend very considerably to the distension of the hæmorrhoidal vessels and the production of piles.

Our ideas of the liver, derived as they generally are from seeing the organ in the dissecting room or on the *post-mortem* table, are frequently quite erroneous, for we are apt to believe it to be a hard, solid, unyielding organ; whereas, on the contrary, a sponge would more nearly represent its behaviour. If we take the liver of an animal, such as a rabbit, which has just been killed, and pass a current of defibrinated blood through it by means of a cannula tied into the portal and another into the hepatic vein, we find that the organ swells up enormously or becomes quite small, in proportion to the pressure with which the blood is driven through it. The rapidity with which this distension and collapse occur is so great as to remind one of the variations in the india-rubber ball of a spray producer. On looking at such an experiment the first thing that strikes us is the question, "If the liver contracts so readily under variations of blood pressure within it, why do we find the size of the liver so constant in man? Why does it not expand and contract as we see it do in the laboratory?" The answer to this is, I think, a very simple one. It is that the blood circulates in the portal vein under a very low pressure indeed, one which is not at all to be compared either with the pressure used in the experiment, or with that which exists normally in the arterial system. But every now and again we do see the liver undergo changes in living men, quite as great, though not so rapid, as in the excised liver of the rabbit of which we have just been speaking. Such changes are especially common in men who suffer from malarial fever, though we see them quite as markedly in the subjects of advanced mitral disease. Such distension of the liver indicates that the

blood cannot flow away through the hepatic vein so quickly as it enters the portal vein, and this condition may either be brought about by too rapid a flow in the portal vein, or obstruction to the circulation either in the liver itself or in the hepatic vein and general venous circulation into which it empties itself. It is quite possible that an augmented entrance of blood into the portal system may be one factor in producing congestion of the liver, but I think it is highly probable that portal congestion is due to obstruction in the liver or beyond it. Nor do I think that obstruction to the flow of blood through the liver necessarily leads to enlargement of the liver, although it may do so. I believe, though it may be difficult to prove, that, either the liver itself, or the portal vein within it, may present an obstacle to the passage of blood, and thus lead to portal congestion without the liver becoming any larger.

On looking at a section of the liver during fasting and digestion, one is struck by the great difference in the size of the cells in these two conditions, the cells being much larger after food. This difference must necessarily lead to a certain amount of compression both of the biliary and venous radicles, and thus hinder to a certain extent the passage of blood through the organ. The walls of the portal vein may also contract and present a certain hindrance. Whenever any portal obstruction occurs it will tend to increase the pressure in the hæmorrhoidal veins, and thus lead to their distension, notwithstanding the fact that the blood in them has another channel of exit. We can readily see that one cause of such an obstruction might be continuous enlargement of the hepatic cells from too abundant feeding, such as gave rise to congestion of the stomach in Alexis St. Martin, as observed by Dr. Beaumont through the fistula in his patient's stomach.

Another cause of portal congestion is, I think, exposure to cold, although whether this acts through the cells of the liver, or through its veins, I am unable to say.

We notice that in the case of internal piles the sphincter ani may affect them either beneficially or injuriously, according to the circumstances under which the pile is to be found. So long as the pile remains inside, the sphincter ani tends to support, and thus to ease it, and will, indeed, aid the circulation of the blood within it. But if the internal pile should become protruded and grasped by the sphincter ani the contraction of the muscle will prevent the

return of the venous blood, will increase distension in the veins, and will render the pile tense and painful.

A somewhat similar action to that of the sphincter ani is said by Verneuil to be exerted by the muscular fibres of the rectum upon the superior hæmorrhoidal veins. These veins pass through little openings, which have been compared to button-holes, in the muscular wall of the rectum. There are two sets of these button-holes at right angles to each other, the first set occurring in the circular and the second in the longitudinal fibres of the rectum. These button-holes, like the sphincter ani in the case of an internal pile, probably have either a beneficial or an injurious action, according to circumstances, upon the hæmorrhoidal veins. It is highly probable, as Mr. Allingham has suggested, that they act as valves tending to support the column of blood in the portal veins when there is congestion of the portal system, as, for example, in mitral disease; but it seems highly probable that they tend also, under other circumstances, to impede the return of blood from hæmorrhoidal veins by too greatly constricting them, just as the sphincter ani does with a protruding pile. Too great a constriction of these fibres would explain the occasional very rapid occurrence of piles, such as we sometimes find after a violent motion of the bowels, especially when this has been brought on by some intestinal irritant, and particularly by such as seem to have a selective action on the lower bowel, like aloes. A similar excessive contraction, due to temperature, may also be the explanation of the well-known frequent occurrence of piles after sitting on a cold stone or on damp grass. It is clear that however strongly the muscular fibres of the rectum contract, they will not cause great obstruction to the return of venous blood through those button-holes if the longitudinal and circular fibres contract with an alternative rhythm as they ought to do, because the contraction of the one set of fibres will be accompanied by relaxation of the others, and the flow of blood through those button-holes will be accelerated rather than hindered by this alternate contraction and relaxation having a kind of pumping action. But it is quite different if either the one or the other set of fibres should contract continuously, and such continuous contraction probably affects the circular fibres during prolonged straining at stool, when the bowel tends to be everted. Accumulation of fæcal matters in the intestine may interfere with the venous return, or may act as a reflex irritant.

We may classify the causes of venous obstruction leading to piles as due (*a*) to portal congestion, and (*b*) to local irritation and contraction of muscular fibres in the rectum itself.

But we have hitherto left out of account two other important factors, viz.:—Dilation of the hæmorrhoidal arteries and local irritation of the veins themselves. Both of these probably play an important part in the causation of piles. The part taken by the arteries frequently becomes evident to the sufferer himself from the throbbing pain felt in the bowels and coincident with the arterial pulse. Local irritation of vessels, both arteries and veins, has frequently the effect of causing them to dilate. When working in Ludwig's laboratory in 1869, I made a number of observations upon the effect of local irritation on arteries and veins. In some of the experiments I made under Ludwig's direction the nerves of a part were all cut through, and in the arteries whose nervous supply had thus been destroyed I noticed that the arterial walls, instead of contracting as they usually do upon irritation, became dilated, and the dilation assumed a somewhat sac-like character, which lasted for a long time after the irritation had been discontinued. I observed a similar occurrence in the veins, but, if I remember aright, the veins tended frequently to become dilated on local irritation, even where the nervous supply had not been destroyed, although it is quite possible that the conditions under which the veins were observed may have partially disturbed their innervation. We cannot, therefore, throw on one side the nervous supply of the hæmorrhoidal veins as of no account in the production of hæmorrhage; on the contrary, it may have a very important action indeed, although we may not be at present able to explain it or to define its limits.

Let us turn now to the conditions which tend to bring on piles. First of all then we have too free living with insufficient exercise, so that the liver, which may be compared to the coal-bunker of the body, has its cells too constantly filled with reserve nutriment and this tends to present a hindrance to the passage of portal blood. Next comes what is usually known as a chill on the liver. What the exact pathology of this is I cannot positively say, but it is a condition which comes on with very great readiness in people who have suffered much from malaria, and in them we find that the liver tends to become larger than usual—sometimes only a little, sometimes very much larger—and at the same time becomes tender to

touch. This condition is frequently associated with loss of appetite, and sometimes with intestinal pains, and a frequent concomitant of it is piles. This condition is brought on in persons subject to malaria with very great ease indeed, and the observations made upon them are most instructive, as showing us how to treat not only such patients but also others who may suffer from the same causes in a less degree. There are four places in such persons which are apt to be affected by a chill. First, the back of the neck; second, the abdomen; third, the shins; and fourth, the feet. The danger of wet feet is universally recognised, and no one wonders when a person gets gastric or intestinal catarrh, or both together, after sitting in wet boots. The danger of cold to the abdomen is almost universally recognised in tropical countries, and in India people will wear many turns of cloth round their middle who have little covering to the rest of their bodies. The danger of chill to the back of the neck is less recognised, but while a cold wind blowing in the face may be braved with impunity, I have seen a cold draught on the back of the neck bring on a fit of ague in a little more than five minutes. The risk of a chill to the shins is still less known, but is very important and is, perhaps, the cause of more unsuspected disturbance of the liver than all the others put together. While the body and feet are warmly clad, people frequently go about wearing short socks and thin merino or silk drawers, which form a very imperfect protection from the cold air which passes up under the trousers. Thus it is that in travelling the legs frequently become chilled. But a chill is got more often still by the person sitting between the door place and fire. As the fire burns briskly in an open grate, the heated air passes in a rapid current up the chimney, and its place must be supplied by fresh air from outside the room. It is quite unusual to find a Tobin's tube or other ventilating apparatus in a room, and consequently the air must come either through crevices in the window or the door. In a well built house the windows fit tightly; the door also fits tightly into its frame on both sides and at the top, but underneath there is usually at least half an inch between the door and the floor, so that it may not rub against the carpet. Underneath the door the cold air comes, and if the fire is burning briskly, there is a strong draught which may be felt by any one who puts the hand to the foot of the door. This cold draught diffuses itself along the floor on its way to the fire-

place, and if any one sits in it his feet and legs are apt to become chilled. He may not be aware of the reason, for neither he nor any one else may be able to feel a draught at the place where he is sitting. Because even though a draught be quite strong at the doorway itself, it becomes too weak to be felt by the hand held close to the floor across the room for a few minutes. Yet it may be quite sufficient to chill the limbs of any one sitting in it for half an hour or longer.

The next cause of piles is local congestion of the rectum by straining at stool. This must be carefully avoided and the patients instructed not to remain long in the closet. There is frequently a tendency to do this when there are piles, even when the bowels move freely, because the piles themselves give a sensation to the patient of something in the rectum, and he strains to get rid of what he believes to be the fæces. The more he strains the worse he gets, until the piles come outside, and then he may think that the bowels are empty. In cases where the motions are very constipated an aperient pill, liquorice powder, cascara, or other simple laxative may be employed to keep the bowels open; or glycerine enemata or suppositories, or an injection of oil or of simple water, may be used.

And here I think it is worth while to give a warning against the water closet as a place where there is great risk of another sort, and which indeed may lead to fatal consequences. As a rule the water closet is destitute of every means of warming it, and people who would never dream of going out of the house into the open air without warm clothing will sit in the closet, which is quite as cold as the external air, not only without any extra covering, but with a considerable portion of their body exposed, and will sometimes remain there for as much as ten minutes, or even more. They thus run a great risk not only of chill to their abdomen, which may lead to portal congestion and piles, but even of getting a chill which may result in pleurisy or pneumonia. This danger is one which ought to be avoided in cases of convalescence from acute diseases, such as influenza. I have been astonished to find so many patients completely neglect what appears to be such a natural precaution. In such cases the patient ought to be obliged to use a commode in his bedroom, or the closet should be warmed by a large paraffin lamp or stove.

Local congestion by excessive straining may be due not to con-

stipation but to diarrhœa. The intestinal condition which gives rise to this may be due to acrid substances which not only irritate the upper part of the bowel and give rise to increased peristalsis, but they may have upon the rectum a double action, namely, an irritant one on the mucous membrane giving rise to straining and sometimes to a feeling of burning; also an irritant action upon the veins and arteries tending to cause dilation and varicosity. Another condition of the fæces which may give rise to local irritation, is a pulpy sticky condition, so that they are with difficulty removed. This condition leads to increased rubbing of the anus with paper in order to cleanse; and if small external piles be already present, the difficulty of cleansing is rendered greater, because the fæcal matters tend to rest in the crevices between the piles and so are removed with difficulty. If printed paper from which the ink comes off be used, the irritation appears to be greater, and the mere continued use of such paper may tend to cause piles even in persons who might otherwise be free from them.

Local congestion of the rectum is increased or brought on by sedentary occupations; for in these, not only does want of exercise retard the flow of blood through the liver, but the local warmth tends to cause distension of the hæmorrhoidal vessels, and bring on piles. When persons have to sit much, they ought either to use a hard wooden chair, or a cane-bottomed chair. If by chance the chairs with which they are provided in the offices they hold are soft and stuffed, they may use a circular cushion with a hole in the middle so as to give a certain amount of ventilation and coolness to the neighbourhood of the anus itself, and such a cushion also eases piles already present by relieving pressure upon them.

There is still another factor to be borne in mind in the causation of piles, and that is the general condition of the patient as affecting the state of his vessels. Gouty people are particularly prone to phlebitis, and in them we find inflammation of the veins of the legs occurring now and again without any apparent reason. But we know as a matter of experience that a glass or two of champagne tends to exacerbate gouty symptoms as a rule, and in some people a glass or two of champagne may bring on an attack of piles. Of course it is possible that the wine here acts partly through the liver by obstructing the circulation in it in the way

already discussed, but it seems highly probable that it has another action as well upon the hæmorrhoidal vessels themselves.

Turning now to the treatment of piles we may consider, first, how we are to keep the liver in such a condition as to maintain a free supply of blood through it. For this purpose we should insist on moderation in cases where we have reason to believe that either the food or the stimulants taken are in excess of the wants of the organism. The occasional administration of small doses of a mercurial purgative followed by a mild saline tends to keep the liver free and to prevent piles, although one may not know the exact *modus operandi* of the mercury upon the liver. Of course the saline ought not to be too violent, or it will tend to cause local congestion and make matters worse. Aloes bears an evil repute on account of its irritant action upon piles, but its effect depends upon the quantity given; and while a large dose of an aloetic pill will almost of a certainty produce rectal irritation, small doses, such as the 1/10th grain of aloin three times a day with each meal, will tend to lessen piles by keeping up a gentle peristaltic action and preventing constipation. My friend Mr. Archer tells me that he has used with invariable success half an ounce of castor oil given to begin with, and followed up by half a drachm every morning as a purgative for a month.

I have already discussed the prevention of portal congestion from chills, but when it has occurred a useful application is a hot water india-rubber bag, with a plush or flannel covering put under the back of the neck, and a similar one over the liver. These tend to restore the equilibrium of the circulation and to lessen portal congestion.

Exercise is useful in keeping the liver free; but this exercise must be of a certain kind. As I have already said, the liver is a very spongy organ, the blood pressure within it is very low, and the pressure under which bile is secreted is also very low. Both blood and bile, therefore, tend to stagnate within it, but this stagnation is lessened by the liver being rhythmically squeezed more or less forcibly between the diaphragm and abdominal muscles. In a person standing or sitting upright or lying on either side, this squeezing action is very slight; in a supine posture it is slightly greater. In ordinary walking it is also very slight, but in walking up a hill, and especially in climbing a mountain, the amount of pressure to which the liver

is subjected is considerable, because the muscles of the abdomen in such exercise are actively contracting, and the movements of the diaphragm during the panting breathing which occurs on exertion are much greater than when a person is quiet. A similar process of squeezing occurs in brisk horse exercise, either trotting or cantering, and thus riding is frequently beneficial for piles, notwithstanding the increased local irritation from contact with the saddle. Another useful exercise is to make the person touch his toes with his fingers without bending his knees several times every morning.

A regular action of the bowels is of the utmost importance in preventing piles, because it tends not only to keep the circulation through the liver free, but prevents straining. The different means of ensuring this regularity of action would require a paper by themselves; but a teaspoonful of compound liquorice powder at night, or confection of senna either alone or with confection of sulphur and confection of pepper, are perhaps amongst the most widely employed of all the laxatives. No doubt the best time ordinarily for emptying the bowels is after breakfast, but if the piles tend to come down much it is better for the patient to get into the way of emptying the bowels every night before going to bed, so that he may secure rest in a recumbent position for several hours. Some patients in whom the piles come down easily spend a day of misery if they are obliged to go to the closet in the morning instead of the evening, because the piles tend to remain down all day and worry them.

The soft unprinted papers which are now commonly sold are a very great improvement upon the ordinary newspapers, but even they sometimes give rise to a good deal of irritation. In cases where the piles are very troublesome it is always well for the patient to wash the anus immediately after a motion. It is sometimes impossible for the patient to go from the closet to his bedroom and wash there, and I have found the easiest way of getting over this difficulty is for him to carry with him to the closet a soft sponge in a small india-rubber bag, an ordinary tobacco pouch is best. If it should be an earth closet, the patient should take the sponge full of water, and after cleansing the anus gently with paper he may thoroughly sponge, and then return the sponge to the bag. The anus may then be dried either with the porous paper or with a small napkin which he carries with him in his

pocket. In the case of a water closet the sponge may be taken dry, and after the closet has been used the plug may be drawn and the sponge dipped in the clean water which then fills the pan and used in the way I have just mentioned. The patient should also take with him to the closet a small bottle of some preparation of hamamelis and some prepared wool. This should be real wool deprived of its fat and not cotton wool. The wool thus prepared is quite absorbent and takes up the hamamelis readily. It differs from the cotton wool in one important particular, for it forms a kind of felt which the cotton does not. A small pledget of the wool about the size of a hazel nut should be dipped in the hamamelis and introduced within the anus, and a similar pledget likewise soaked in the hamamelis should be introduced so far within the anus that a few fibres of it at least are caught by the sphincter. The external pledget soon becomes felted together into a regular pad fitting completely to the anus, and being retained by the few fibres caught by the sphincter it will remain there for twenty-four hours, while a similar pad of cotton wool might not remain as many minutes. This wool pad not only keeps the hamamelis in constant contact with the piles, but also affords a certain amount of mechanical support. In patients suffering from piles we frequently notice an almost involuntary tendency to sit on the corner of a table or on the arm of a chair, or to put the hand behind and press upon the anus from time to time; but the woollen pad by affording a constant support tends to lessen the necessity for pressure in any of these ways. Where the piles are chiefly internal the hamamelis may be applied in the dose of half a drachm to a drachm either diluted with water or, as is sometimes preferable, undiluted, by injecting it within the anus by means of a glycerine syringe. The success of this treatment in stopping hæmorrhage from piles is really extraordinary; within a week I have stopped the hæmorrhage from piles which were bleeding so profusely that a colleague thought that an operation would be necessary. But not only does the hamamelis stop hæmorrhage, it lessens the uncomfortable weight and aching pain which so frequently accompany piles, especially when they do not bleed, and it will even greatly lessen or remove the pain which occurs in piles when they become inflamed. I have tried various preparations of hamamelis, but I have not found either the tincture or the liquid extract, both of which are to be found among the recent

additions to the Pharmacopœia, nearly so satisfactory as some of the proprietary preparations.

The patient requires to be carefully instructed in the mode of using it, otherwise disappointment may ensue. Some time ago a lady who was passing through London on her way to the Continent was seized with a sharp attack of piles. I was asked to see her at an hotel, but not being able to go for a couple of hours, I hastily wrote down a prescription for hamamelis and gave it to the maid with, as I thought, definite instructions how to apply it. On going to the lady two hours afterwards I found that she had used the whole bottle, but with no relief whatever, nor was this to be wondered at, for the piles were internal, and the hamamelis had only been used externally. So satisfactory have I found hamamelis that I do not often now employ ointments.

In obstinate cases of piles great relief is afforded by the anal pad. The simplest is one of india-rubber with elastic straps to hold it in place, but it does not give, I think, quite the same relief as one in which the pad is pressed against the anus by a spring attached to a metal girdle which passes round the loins.

Before concluding this paper I may mention another affection which frequently goes along with piles and is most annoying, namely, pruritis and eczema round the anus. Both of these affections may be lessened by the simple plan of applying Eau de Cologne to the itching surface with a small sponge or a pad of cotton wool. If the skin be at all tender, undiluted Eau de Cologne gives rise to intense burning pain, but this may be prevented by diluting the spirit before application. The diluted spirit does not have such a strong and permanent action in lessening the itching as the pure spirit, and where the itching is at all great the pure spirit may be used notwithstanding the pain it causes, for it converts the intolerable itching into a severe smart, and this may be relieved by diligently fanning the part till the spirit evaporates.

I have not attempted to discuss all the methods of treatment. I have rather brought forward some which I have found practically exceedingly useful, and which are, I think, at least in their details, not so widely known as they deserve. I am quite conscious how trivial they are, but the number of cases in which piles occur give an importance to any useful method of treatment, however trivial it may be in itself, and this must be my excuse for bringing the subject before the Society.

Mr. CRIPPS drew a sharp line of distinction between internal and external hæmorrhoids, both in regard to pathology and treatment. The external, he thought, were far the more common, and were totally unconnected with disorder of the liver. Experimentally he had been quite unable to inject the hæmorrhoidal veins from the iliacs. He recognised two varieties: the one, in which, as a result of straining or accident, a small vein burst and gave rise to a thrombus in the folds of cellular tissue around the anus; the other, which occurred more frequently, was due to the development of œdematous folds of skin in the same position, which were tender and painful and often due to the presence of a fissure. This, which was really the primary lesion, resulted from constipation or from cracking of the epithelium, as occurred in the lips in cold weather. Internal hæmorrhoids became obvious when inflamed, or prolapsed and strangulated, but attention was still more frequently called to them by hæmorrhage.

Dr. LAZARUS-BARLOW thought that the influence of the liver in the causation of piles was greatly over-estimated, and even doubted whether that organ was ever the real cause. He instanced advanced lardaceous change, cancer, and pyelo-phlebitis, as conditions under which the portal circulation was, without doubt, greatly impeded, and yet piles were not especially common or severe. With regard to congestion of the liver, he doubted the possibility of its safe diagnosis, except in cases of morbus cordis, and compared the frequency of mitral disease and its hepatic complication in young children with the marked infrequency of piles at an early age. Nevertheless, in cases of cirrhosis the question was not so clear; the concomitant varicose condition of the veins around the lower end of the œsophagus, forming a link between the portal and general venous system, seemed to support the view of hepatic origin of piles; but in cases of cirrhosis dilated veins in the pharynx and stigmata on the face were also common, and they could not have the same explanation. Piles were very commonly met with in pregnancy, but though the source of pressure was the same, the vagina, though quite as well supplied with veins as the rectum, never showed any appearance comparable to piles. Lastly, he held that the result of surgical operation was to negative the hepatic vein, as removal of the anastomoses of the veins, on that view, could not but invariably leave the patient in a worse condition than before, which was far from being the case. He regarded the affection as dependent upon a loose state of the mucous membrane, and as predisposed to by prolonged straining at stool, which he thought was very common in childhood.

Mr. GOODSALL also exonerated the liver from any share in the production of piles. He thought the principal factors were heredity, constipation, and any cause of local congestion. In young women they appeared with remarkable constancy on the right side of the anus near the perineum. Hæmorrhoids were more frequent in people with lax tissues, who were also the subjects of varicose veins and hernia. Sedentary occupations favoured the malady.

Mr. PEARCE GOULD thought that the most notable outcome of the discussion was the evidence it afforded of the want of a new and better nomenclature for the diseases commonly called "piles." An "attack of the piles" had that evening been defined as four entirely different conditions:—(1) As an outburst of hæmorrhage, (2) as prolapse and strangulation of a part of the mucous lining of the rectum, (3) as the subcutaneous rupture of an anal vein forming a so-called thrombus, and (4) as an

inflamed fold of skin around the anus from infection through an abrasion or crack of the mucous lining of the anus. These four conditions differed very widely in their nature, and only confusion resulted when they were spoken of as one disease—"piles." Before the pathology and treatment of this group of affections can be profitably discussed, it must be split up into its component members. As to the point chiefly laid stress upon by Dr. Lauder Brunton, his experience led him to attach most importance to local causes of "piles," and especially to the effect of constipation and the passage of large and hard motions. This led to straining and also to bruising, abrading, stretching, and displacing the parts about the anus. As one evidence of this he instanced the signal relief afforded in many cases of "piles" by the daily use of an enema.

Dr. BRUNTON, in reply, said he was glad he had introduced the subject, as it was evidently one which required a good deal more discussion, but he regretted not having given more time to making his views clear. He regarded the liver and variations in its circulation as being only one of many factors in the production of hæmorrhoids. There was no doubt local dilatation of arteries as well as veins assisted, by straining, local irritation and abrasion. The evidence of congestion in the liver was similar to that of congestion elsewhere—enlargement and tenderness. The liver was liable to vary in size greatly and with remarkable rapidity.

March 14th, 1892.

CERTAIN QUESTIONS ON THE TREATMENT OF DIABETES.

By CHAS. H. RALFE, M.D. Camb., F.R.C.P. Lond.

THE questions which I propose submitting for consideration are—1. In cases of confirmed* diabetes running a protracted course, may any relaxations from the usual dietetic restrictions be permitted? 2. In such cases, at what period of the disease should opium or its derivatives be commenced, and how far may they be pushed? Besides these, I had in view a third—viz., What other measures, in addition to diet and opium, have been found useful in checking the disease or in relieving special symptoms? But on consideration, when I came to write this paper, I found that the addition of this third question would make the communication too lengthy; besides which, I thought it would distract discussion from the other two, which are, to my mind, the most important. At first sight, it may be thought

* Cases of transient and gouty glycosuria and intermittent forms of diabetes, in which assimilation is for a time restored, are not considered in this paper.

that these questions are superfluous, especially in this country, where the insistence on the maintenance of a rigid dietary, combined with the administration of opium or its derivatives, has been taught in our schools and endorsed in our text-books. But of late, views have sprung up which have had a tendency to impair this teaching, especially as regards the necessity for maintaining a strict adherence to a proteid diet as the essential point in the treatment of the disease. This laxity with respect to a canon law first originated, no doubt, with the introduction of the so-called "skim-milk cure," and as at that time the distinction between diabetes and transient glycosuria was not clearly defined, several cases of the latter were cured which were supposed to have been of the nature of true diabetes; and though Dr. Pavy promptly disclosed the fallacy, and again placed the ban against milk in confirmed cases of diabetes, still there has remained a leaven of the heresy among many practitioners, not merely in not excluding milk from the dietary, but even in ordering it in very considerable amounts. But milk is not the only article that has crept in unawares into the fold of a pure dietary, and many of the Continental physicians now permit a dietary which can no longer be called restricted. Thus Dr. Seegen* permits a small quantity of wheaten bread and subacid fruits, such as strawberries, &c.; Dr. Coiquard† and Dr. Dujardin-Beaumetz‡ allow mashed potatoes instead of gluten bread; whilst in America, in a well-known work on practical medicine,§ the diet table drawn up by Dr. Austin Flint|| permits of asparagus, gooseberries, cherries, plums, and strawberries. It is true, no doubt, that those who permit these relaxations do not question the theoretic considerations on which the absolute restricted dietary is founded; they only deny the possibility of carrying it out to its full extent, and point out the direction in which relaxation may be least harmful to the patient. The point, therefore, I would draw attention to is whether such relaxation should be forbidden as aggravating the disease, or forced on us as a compromise.

* International Medical Congress, Berlin, 1890; *vide* report in 'The Lancet,' September 15th, 1890.

† 'L'Union Médicale,' November 20th, 1886.

‡ 'Bull. Gén. de Thérap.,' 1886, cxi, p. 385.

§ 'Pepper's System of Practical Medicine,' vol. ii, art. Diabetes, 1885.

|| 'New York Medical Journal,' May 24th, 1884.

With regard to the second question, little information exists ; for though the authorities on this subject are eloquent on the effects opium and its derivatives have on the course of the disease, few, if any, rules are given for guidance.

The answer to these questions will, of course, greatly depend upon our clinical experience, and the views we have formed as to the nature and progress of the cases that have come under our observation. Most authorities now recognise two forms of confirmed diabetes, which they classify as—(1) neurogenic, usually coming on suddenly as the result of some nervous shock, and running a more or less acute course ; (2) constitutional, gradually supervening as the result of some taint—gouty, rheumatic, syphilitic, or tubercular—the progress of which is usually protracted. But of late I have accepted a distinction between the two forms which I think indicates more closely their relationship and better represents their clinical features than the above-mentioned classification, which is based only on etiological conditions, and which I would describe as : 1. An alimentary diabetes, which can at first be entirely controlled by diet ; but gradually this control is lost, and sugar reappears in the urine in spite of all restriction. This form is most commonly associated with a constitutional taint of some kind, though it sometimes, but less frequently, is found in cases of neurogenic origin. 2. General diabetes, in which, from the earliest onset, a restricted diet fails to remove entirely the sugar from the urine, whilst the glycosuria is out of all proportion to the food (of all kinds) ingested. This form is common in cases of neurogenic origin, and is specially characteristic of pancreatic diabetes. Whence comes the sugar in urine which is not derived from the starchy and saccharine materials of the food has always been a problem. That it is not altogether furnished by the metabolism of the proteid elements of the food—which was formerly supposed—is shown by the fact that it is often in considerable excess of what could be furnished by the proteid diet the patient was on ; besides, as I have observed in carefully dieted cases, hardly any reduction, certainly not at all proportionately, follows a diminution of the proteid food—a fact which points to the source of the supply being from the tissues themselves ; and, indeed, we now know that other tissues besides the hepatic furnish glycogen. What then probably happens in the two forms of diabetes we are considering is, that in the milder and alimentary form the

power of assimilating starch and sugar is only lost as far as the liver is concerned ; but after a time, perhaps from the passage of sugar from the liver into the circulation, the tissues lose the power of transforming glycogen into energy or storing it up as fat, or, as some are now endeavouring to show, the blood loses its glycolytic or sugar-destroying power, owing to the absence of a ferment which is said to be normally furnished chiefly by the pancreas, but also by other glands. It is then that the diabetes may be said to become general. Out of a total of sugar excreted by the urine, a portion still remains removable by abstinence from starchy and saccharine food, but as the case progresses the proportion between this removable sugar and that which is not affected by a strict diet becomes more and more marked. Thus in a mild case in an early stage the total amount of sugar passed in the twenty-four hours was 150 grammes, which on strict diet sank to 30 grammes, or 5 to 1 ; whereas, in a severe case, one month before death, the total amount of sugar was 320 grammes on a mixed diet, which was reduced by strict diet 190 grammes, or a proportion of 1 to 1.6. In short, the difference between the removable and non-removable sugar in a case of diabetes is one of considerable value for the purpose of prognosis, for any sudden increase in the proportion of the latter, though there may not be any actual increase in the total of sugar excreted, is always indicative of an exacerbation of the disease.

Rollo was the first to point out the extreme susceptibility of cases of diabetes, in which the sugar excretion had been stopped by diet, to relapse on very slight and very inadequate resumption of sugar. "Thus, half a biscuit, such as sold by confectioners at three a penny, has," he says, "brought back the disorder in full force," and my own observations have convinced me of the fact that in alimentary diabetes any increase of sugar ingested is always followed by an excretion of sugar, often above that amount. On the other hand, in general diabetes, although it is manifest the assimilative processes are lowered by the persistent presence of sugar in the blood, still the proportionate effect on the excretion of sugar is not so marked as in the purely alimentary form. Thus in a case in which the proportion of removable sugar was as 5 to 1 of non-removable, the resumption of mixed diet caused an immediate increase of the total sugar, amounting to 80 per cent. ; whilst in a case in which the proportion of removable sugar was

as 1 to 2 of non-removable, the resumption of the same dietary only caused a rise of 27 per cent. At first this would seem to tell in favour of a partial toleration of a less restricted diet in protracted cases of diabetes, especially when the proportion of non-removable sugar is high; but further observations show that though the increase per cent. in the amount of sugar in these cases is not so large as in the alimentary ones, still the resumption of saccharine food, even in a limited extent, acts adversely against the assimilative processes in the body, as is shown by the fact that generally when strict diet is again resumed, we find the proportion between the sugar removable by diet to be decreased; whereas the non-removable sugar is found to be increased. Thus, for instance, when before the resumption of a mixed diet the proportion of removable sugar was 3 to 1·5 of non-removable, after three weeks of mixed diet, on the return to an absolutely restricted diet, the proportion became 2 to 2·5, showing that persistent supply of saccharine food had further lowered the assimilative processes in the body. This fact, therefore, seems to tell conclusively against any relaxation of the restricted dietary in this form of diabetes; whilst the extreme sensitiveness to the minutest particle of starch and saccharine food exhibited when the glycosuria is still controlled by absolute restriction of the diet tells equally against its resumption in this stage of the disorder.

The next consideration is whether the advantages gained by a strict adherence to an absolute diet of proteid substances by diminishing the amount of sugar in the blood, and so checking the tendency to a further lowering of the assimilative processes in the body and controlling the extreme diuresis, may not be gained at too great an expense to the patient's well-being, and that some benefit may be derived in other directions by permitting a slight relaxation from too rigid a proteid dietary, and whether its too long continuance is not in itself a danger by causing the formation in excess of bodies such as the morbid products of proteid metabolism. This is a question that requires further investigation before a decision can be given, but there are certain facts already ascertained which certainly seem to point in that direction. Dr. Wright, in his Grocers' Research Scholarship lecture,* stated that in the artificially induced diabetes of an animal kept entirely without food the same morbid products of proteid meta-

* See 'The Lancet' of February 27th and March 5th, 1892.

bolism were found in the urine as are found in human diabetes, which, as the animal was in a state of inanition, must have been furnished by the imperfect metabolism of its proteid tissues. To this it may be answered that certain tribes live almost exclusively on meat from year's end to year's end; as do hunters for very long periods at a time without appearing to be in any way injuriously affected by diet. But the powers of assimilation of healthy active savages or vigorous hunters is very different from the feeble diabetic. In scurvy—a disease as I have endeavoured to show brought about by the withdrawal of alkaline salts and the increase of acid salts—so long as the patient remains vigorous, active, and cheerful, the disease is kept at bay; but the moment the vital powers are lowered from exposure, fatigue, or anxiety, then all the consequences of the diminished alkalinity of his blood are experienced. So in diabetes, though there is no actual withdrawal of the alkaline salts, there is with the flesh diet a positive entrance of an increased amount of acid salts into the body. These possibly at an early period of the disease, whilst the patient is still fairly vigorous, are eliminated by the kidneys, but when the bodily powers begin to fail, they accumulate to a dangerous extent. Added to this is the fact that with increased feebleness the power of digesting proteid material is lessened, and consequently the risk of the formation of toxic bodies in the intestine is increased.

Admitting, then, the risks attendant on a proteid diet, I would ask, are the relaxations proposed calculated to obviate them? I think not. They may satisfy a craving or help a jaded appetite, but, from a careful consideration of a very fair number of cases, I can confidently assert that the admission of any article containing starch or sugar is always followed by an increase of the disorder, which if persisted in becomes a definite advance. This intolerance of any partial resumption of saccharine material is more marked in some patients than in others, and some more quickly rally from an indiscretion, whilst some forms of sugar are better tolerated than others. Still, the fact remains that any article containing sugar ingested increases the glycosuria and diuresis, and further lowers the assimilative processes in the body. Neither a modicum of wheaten bread nor of mashed potatoes, or, what is a more reasonable suggestion, the use of subacid fruits, is sufficient to counteract the danger arising from the morbid products

of a defective proteid metabolism, which is a danger incidental to the disease itself; whilst they are decidedly hurtful in other directions. To obviate this risk other measures are called for, which cannot be discussed fully now, and which are indicated by improving and maintaining the patient's strength by every hygienic and therapeutic means at our disposal. Two measures, however, I may briefly touch upon. The one is in the regulation of the amount of proteid food as the assimilative powers flag. There is very little doubt that diabetic patients are often encouraged to eat more animal food than they require or can assimilate; and I have seen the greatest improvement follow a reduction in the amount of meat, as also in a change from beef and mutton to fish, poultry, and such light food as tripe, calf's head, &c. A Paris physician of some eminence called attention to this point some years since, and stated that during the siege of Paris he noticed that his diabetic patients made considerable improvement, which he attributed to their having their flesh diet greatly curtailed. The importance of taking plenty of green vegetable food should be insisted on, to say nothing of watercress and other green salads. The second measure to improve assimilation and promote metabolism is massage, both general and over the abdominal viscera. When thoroughly carried out I have seen it effect a wonderful improvement in the patient's condition, in the restoration of appetite, improved digestion, relief of constipation, and a general increase of vigour.

With regard to the second question, I fear but little time is left for its full consideration on the present occasion, but as it is rather to elicit information than to impart any fresh facts, it will not take me long. It is strange, indeed, that, with a drug which is acknowledged as the only reliable therapeutic agent available for the treatment of the disease, no definite rules have been formulated with regard to its administration. Amongst the patients who come to me who have already been under treatment by their family attendant, I find a certain number who have already been ordered opium or one of its derivatives, others who have only been restricted as to diet. On looking over the reports of published cases, I find the same uncertainty of opinion; some relying only on diet combined with some other form of treatment, others in which both diet and opium have been administered. There is therefore no unanimity of opinion as regards the time

when treatment by the drug should be commenced. Some give it quite early ; some wait till the disease is no longer removable by diet alone ; whilst others postpone its administration till quite a late stage, or only employ it when complications, such as boils, carbuncle, or gangrene occur. Some give it freely ; others employ it sparingly.

With regard to the mode of administration, till Dr. Mitchell Bruce's instructive paper in the 'Practitioner,' 1887, it was not demonstrated experimentally that subcutaneous injection of the drug was much less effective in restraining the glycosuria than when taken by the mouth. Even up to the present time it is not decided whether it is best to administer the drug when the stomach is empty, or during the process of digestion, when the portal circulation is in full activity. It is not conclusively determined whether opium in its crude form, or one of its derivatives, is best employed, or whether a combination is not most effective. Lastly, there is no consensus of opinion on the very important point how far we are justified in pushing the drug when the disease is prolonged, or when a fresh exacerbation occurs.

My answer to these inquiries must be brief ; but I hope the conclusions I have drawn from observation of cases under my notice will be found to coincide with those of others, and will be strengthened by the experience of those present to-night.

1. With regard to the period when opium or its derivatives should be commenced, I have come to the conclusion that whilst the glycosuria can be completely removed by diet the drug should not be administered, for this reason : that when it has once been administered, and then left off, on resuming it a much greater quantity is required to effect the same result than if even opiates had been continued throughout. This is in accordance with Dr. Mitchell Bruce's experience, for in one of his cases, quoted in the paper already alluded to in the 'Practitioner,' in which the dose of morphia was reduced for a time, it was found that on again resuming the full dose it was necessary to increase it from $4\frac{3}{4}$ grains of morphia to 7 grains before the same effect was induced. And here I would touch, parenthetically, on the danger of suddenly reducing the dose of this drug when it has been administered for some time, and especially in advanced cases. In two cases in which this has been attempted fatal results followed so shortly after that there could be no mistaking the cause. No

hesitation, however, should be felt in commencing treatment by this drug as soon as the sugar fails entirely to disappear from the urine by mere restriction, as it is then that its power in maintaining the assimilative processes in the body is best exercised.

2. With regard to its mode of administration, the most decided results follow in diabetes when the drug is administered by the mouth, so that any other method need not be entertained. With respect, however, to the most effective period for administration, whether immediately before meals, so that it may quickly enter the portal circulation, or during digestion, when the assimilative processes are most active, I have found that administration about an hour after a meal has a greater effect in restraining diuresis than when taken on an empty stomach, that not much difference is effected on the sugar excretion; but that the dose taken shortly after food has one great advantage, especially when large doses of the drug are taken, of not disordering the stomach, or of causing nausea or impairing digestion as when taken fasting.

3. As to the best preparation of the drug. Solid opium, which was entirely relied on before the introduction of codeine by Dr. Pavy, has fallen into discredit, as I think undeservedly, and that alkaloid has almost entirely replaced it. But Dr. Mitchell Bruce has pointed out that morphine acts equally as well as codeine as a sugar-restraining agent, and, indeed, is more active and more powerful, whilst quite as safe. Whilst endorsing all Dr. Mitchell Bruce says regarding the value of morphine in diabetes, and preferring it to codeine, on account of its greater uniformity of action, I have come to the conclusion that the full benefit of the drug in diabetes is not obtained unless some of its other derivatives are combined with the alkaloids. Though both codeine and morphine restrain the sugar excretion and the diuresis, still patients on whom the test has been made inform me that they experience a greater satisfaction when some preparation of crude opium has been added to either alkaloid. This can be done by combining liquor opii with acetate of morphia in solution, increasing or diminishing one or the other as circumstances seem to require; or else employing some of those extra-pharmacopœial preparations which contain morphia as well as some other derivative of opium. Besides, I may say that diabetics often exhibit individual peculiarities as regards the different preparations of opium, which ought to be studied. In one case (a lady) codeine and morphine were tried

on several occasions, and had to be given up on account of the headache and giddiness which were produced. Solid opium in the form of compound soap pill was, however, taken without any discomfort.

4. Lastly, in fixing the dose, at first it is important to find out the patient's own capacity for the drug, which varies greatly in different individuals, and discouragement should not be felt if the patient complains, or is in any way made uncomfortable on first commencing opium or its preparations. Each patient has his proper dose, and as soon as that is ascertained the full benefit of the drug will be felt. The real difficulty lies in determining the right time for increasing the dose, and when this becomes large to decide whether we should continue to increase it further. As a rule, we err, I think, on the side of too much caution, and often fail to obtain the full advantage of the effects of the drug by holding our hands when a certain effect has been produced. As soon, therefore, as the glycosuria ceases to be absolutely controlled by dietetic restrictions, I venture to suggest that opium should not only be given in doses that sensibly affect the excretion of the sugar, but should be increased till either they entirely control the glycosuria, or no further reduction in the amount of sugar passed is obtained on increasing the dose. When this point is reached, we should recognise that the drug had been pushed to its fullest sugar-restraining capacity, and that the dose need not be increased till a further exacerbation of the disease takes place, always assigning as a limit to its increased use the point at which it ceased any longer to affect sugar excretion. How far and how long the use of opium or its derivatives may be carried without causing injury to the patient there is little means of saying. Diabetics are, as is well known, extremely tolerant of the drug, and it is impossible to judge of them, as regards the effects of opium, as is possible in the case of morphia *habitués*. Nor is it possible to ascertain what are the maximum doses that have been reached. Dr. Richardson, in a paper contributed to the Medical Society on the morphia habit,* mentions the case of an elderly patient, a medical man suffering from diabetes, who for a period of eleven years took from 1 to 3 grains of morphia daily. Ultimately this patient died from what might be called premature old age. He was fairly happy up to a few weeks of his death, and Dr. Richardson

* 'Transactions,' vol. vii, 1885.

had every reason to believe that his comparatively easy life was considerably prolonged by the morphine. Dr. Mitchell Bruce, in the paper already alluded to, mentions 7 grains of morphine as the maximum dose in one case, and $6\frac{1}{4}$ grains in another, whilst under his care in the hospital. I have at the present time a patient who for two years has taken $3\frac{1}{2}$ grains of morphine every day, having previously taken smaller doses. This patient has maintained his general health remarkably well: he is active, cheerful, and follows his occupation to his own and every one else's satisfaction, whilst the drug does not seem to have any of its usual effects—he is not drowsy or constipated; nor does he crave for the drug. For two years this dose has sufficed without any further increase being required; lately, however, the diuresis has increased, as well as the amount of sugar, and an augmentation of the dose must shortly be resorted to. The fact, however, that there has been this pause in the disease shows that opium, when given in effectual doses, does not need that these doses should be frequently added to. In this case, at the last increase, the dose of morphine was increased till no further impression was made on the glycosuria, which fell to about 50 grammes in the twenty-four hours, and has fluctuated between that and 80 grammes during the period mentioned. I have also given morphine to the amount of 6 grains in the day without observing any ill effects; but this quantity was only reached towards the end of the case, the longest period not being more than three months. So long, however, as opium or its derivatives affect a reduction in the excretion of sugar, I believe we may safely increase the dose; but as soon as the point is reached, and this frequently happens towards the close of the case, at which no further reduction is possible, and the sugar excretion rapidly gains ground in spite of dietetic restriction and the administration of opium, then I think it wise not to attempt any further increase.

I have thus, though I feel in a very incomplete manner, endeavoured to answer according to my own experience and observations the two questions which I proposed at the commencement of this paper for consideration, and trust they may be in accord with the general opinion of the profession.

Dr. PAVY, having congratulated the author on the excellence of his paper, said that the views put forward did not exactly correspond with his own. He would lay more stress upon dietetic measures than Dr. Ralfe

appeared to do. He regarded diabetes as essentially the faulty assimilation of the carbohydrate elements of food. The fact should not be lost sight of that glycogen and sugar were universal constituents of the body. Carbohydrate material might be locked up in proteid compounds, such as the glucosides. Mucin, which existed extensively throughout the body, had comparatively recently been demonstrated to be a glucoside, a complex proteid molecule consisting of a simpler proteid in combination with a carbohydrate. As he looked upon all diabetes as being due to defective assimilation of carbohydrate matter, he could not follow the author's classification. The statement often made, that the liver was more saccharine than other organs of the body, was not true, looked at from the point of view of the condition existing during life; nor was it true that the blood of the portal vein contained less sugar than that of the hepatic vein. He had performed a great number of experiments with a view to determine this point, and he found that the blood of the portal vein contained ordinarily so much more sugar than did that of any other vein in the system, that if blood of the same quality as the portal blood circulated through the body generally it would certainly invariably give rise to diabetes. The amount of sugar found in the urine was always proportionate to the amount of sugar at the time in the blood. A trace of sugar could always be found existing in normal urine, which corresponded precisely with the amount found existing in the general circulation. A deviation in the amount of sugar in the urine would indicate a deviation of the blood in this respect from its natural state, and in proportion to its deviation from this natural state would be the impairment of the health of the patient. If his views were correct, it followed that the first object in treatment should be to bring back the blood to its natural state. In some cases there was actually a return of assimilative power, but this would never come about if the urine were permitted to contain a large quantity of sugar. His habit with those patients whose assimilative power was gradually being restored was to give them some carbohydrate, at first in very small quantities, and then gradually increase it, taking the urine as a guide, and keeping it free from sugar. The kind of carbohydrate material administered was perhaps not of much moment, but it should be remembered that substances like cane sugar were more pernicious than the quickly absorbable starches. Given a patient under diet in whom the assimilative power was being restored, experience had shown that whilst previously under the restricted diet weight had been gained, the patient now began to lose, and continued to do so if carbohydrates were altogether withheld. He was able to take this as an indication that the time had arrived for trying the administration of a little ordinary bread, and if such administration were unattended with the passage of sugar the patient would immediately gain weight again. It was curious that in cases of the favourable class, but in which assimilative power did not become restored, the restricted diet might be continued for years with the perfect maintenance of weight, strength, and health.

Dr. LAUDER BRUNTON was glad to hear that Dr. Pavy believed there was a certain percentage of sugar in ordinary healthy urine. For his own purposes he had divided these cases into the true diabetics and the gouty glycosurics; the former were usually thin nervous people, and in them the disease began early in life and ran a rapidly fatal course. In the second category the patients were usually of middle age, full habit, robust frame, and red face. The disease usually yielded readily to diet and exercise, and when the sugar went from the urine it was generally replaced by oxalates

or uric acid. He referred to the case of a lady aged 85, who for thirty years had passed a quantity of sugar in her urine, but she was still healthy and strong. Though there were these two distinct classes, yet he had often met with mixed cases. In the second variety he had found the administration of salicylate of soda of more benefit than that of opium. He referred to an article which he wrote many years ago in 'Reynolds's System of Medicine,' in which he rather threw cold water on the skim-milk treatment of diabetes. He desired now to correct that criticism, and to state that he had found this treatment very useful, especially in cases in which the glycosuria was combined with albuminuria; in such instances it gave sometimes very good results indeed. He advocated as an article of diet useful to diabetic patients the *pommes de terre frites* as cooked in France. They were not really fried, but the potatoes were cut in very thin slices, thrown into boiling oil, which browned them at once, and then they were taken out and dried. As a rule English cooks, in attempting to make this dish, did not use enough oil, and they did not make it hot enough. They served excellently to relieve the monotony of a purely proteid diet. In hospital practice he had been accustomed to prescribe for diabetic patients a draught containing iron and morphine, which he had found very useful in those cases in which it was impossible to carry out a rigid system of diet. But more than one case had ended fatally under this treatment, although the morphine only amounted to three-sixteenths of a grain in the twenty-four hours. He had now made it a rule never to administer morphine if the aceto-acetic reaction with perchloride of iron were present in the urine.

Dr. MITCHELL BRUCE agreed in the necessity of endeavouring to lay down definite rules to follow in particular cases. First, as to the time when opium should be commenced, he did not think it necessary to begin to administer opiates until the dietetic treatment had been exhausted. With respect to the form of drug which should be chosen, no doubt many cases were better treated with opium than with the pure alkaloids; but he himself had selected morphine because it was a definite substance, and its dose could be so easily regulated; and of the salts he had chosen the acetate in order that he might administer it subcutaneously if he wished to. It should not be given sparingly if all were going well, but it was unwise to push it to extremities in order to rid the urine of the last small quantity of sugar which it might contain; it was most difficult to get rid of the latter, and it was foolish to try to do it by means of an increase of the remedy.

Dr. ALLCHIN (Vice-President, in the Chair), in thanking Dr. Ralfe for his able and interesting paper, expressed the opinion that in the class of cases which was the subject of the communication, viz., those of a chronic character in elderly people, that an absolutely restricted diet was very frequently undesirable, as it was often difficult or impossible of attainment, and that a considerable licence might be permitted with advantage under medical advice. He also entered a protest against making the degree of glycosuria the sole indication for the treatment, whether dietetic or medicinal, of such patients. He knew of individuals who were passing a very considerable amount of sugar daily whose general health was excellent notwithstanding that they indulged very considerably in starchy foods and took no drugs. He regarded the amount of sugar as only *one* of the symptoms on which a plan of treatment was to be determined, and whenever the general health was good and the body weight constant, so long as the amount of sugar varied little from day to day, great laxity of diet

might be allowed, and was even in many cases imperatively called for. The glycosuria was to be regarded as an evidence of perverted tissue metabolism, and when occurring in association with another condition of morbid tissue change, viz., gout, in elderly diabetics might exist to a considerable degree without any serious disturbance of general health. These remarks of course did not apply to acute cases, nor to diabetes in young persons.

Mr. G. STILLINGFLEET JOHNSON wished to make a few remarks on one point which had been touched upon by Dr. Pavy in the course of his comments upon Dr. Ralfe's paper, viz., the question of the presence of sugar in normal human urine. Dr. Pavy had expressed the opinion that sugar is invariably present in normal human urine. Mr. G. S. Johnson believed that it was invariably absent, and that the presence of sugar in urine, even in small quantities, was evidence of diseased action. Six years ago Mr. Johnson performed a series of careful experiments in order to discover the nature of the reducing agents which are invariably present in normal human urine, and he was led to the conclusion that the reducing action of that secretion may be accounted for by the uric acid and the kreatinin which it contains. Mr. Johnson attributed the low reducing power of kreatinin as hitherto obtained to isomeric changes produced in the base during the process of extraction from the urine, for the kreatinin prepared by himself is a very powerful reducing agent.

Dr. RALFE, in reply, thought that they were all very much in touch upon the question of diet. Dr. Pavy, however, had somewhat mistaken the drift of the paper, which was to insist strongly on dietetic measures, as also to point out that the glycosuria might be furnished by the carbohydrate element in proteid tissues. His paper had no reference to cases in which the assimilative power was temporarily restored, though he agreed that there were so many cases of all grades that it was exceedingly difficult to classify them. Of the opium preparations, he relied upon morphine as being a more definite substance than codeia, though he often combined it with advantage to some other preparation of the drug.

March 28th, 1892.

RAPID DILATATION OF THE UTERUS FOR DIAGNOSIS AND TREATMENT IN CASES OF UTERINE HÆMORRHAGE.

By AMAND ROUTH, M.D., B.S.

EXCESSIVE or prolonged hæmorrhage from the uterus is so commonly under treatment that a discussion on the means of ascertaining its causes, and their appropriate treatment, seems likely to be useful, especially if those who have had a wider experience than myself will give the benefit of their views.

It is intended here to deal solely with exploratory dilatations of the uterus for *hæmorrhage*, omitting therefore all cases of dilatation for dysmenorrhœa, or chronic endometritis without hæmorrhage, or those rare cases where it may be necessary to dilate the uterus to admit the finger with a view to break down retro-uterine adhesions after Schultze's method.

Necessarily also, cases of uterine hæmorrhage due to intra-uterine causes discoverable and removable without mechanical dilatation are excluded. Among such may be named hæmorrhage due to an intra-uterine polypus already extruded into the vagina, or *post-partum* hæmorrhage where the uterus is already sufficiently dilated by the passage of the fœtus or ovum.

If then a woman comes for treatment, one of whose chief symptoms is menorrhagia or metrorrhagia, enquiries would be made as to constitutional causes, and a vaginal examination would be made, unless for the time being contra-indicated by virginity or youth. A vaginal examination should certainly be made if hæmorrhage occur after the menopause.

Possibly some obvious causes would then be discoverable, such as cancer or adenoma of cervix, adhesive ulcerative vaginitis, severe erosion of cervix, ulcerations of vagina from retained foreign body, an extruded fibroid polypus, a cervical mucous polypus, ulcerating procidentia uteri, or inversion of uterus. The possibility of molar pregnancy, or endometritis of the gravid uterus, would also not be forgotten.

A bimanual examination would then be made, and the uterus, perhaps, found to be enlarged by subinvolution, or fibroid tumour, or there might be some dilatation of the tubes, or some peri-uterine condition, the existence of which would forbid further local interference. Subinvolution with backward displacement of the womb, as a frequent cause of hæmorrhage after labour or abortion, has been recently statistically proved by Dr. Herman.

If no obvious cause were now made out, and pregnancy could be excluded, a careful examination of the uterine cavity by the sound would ascertain its size and shape, and the roughness or otherwise of its lining membrane.

If the uterus be not much enlarged, and the hæmorrhage not profuse or frequent, a fair trial of constitutional remedies may be made. If the hæmorrhage may be reasonably assumed to be due to a chronic non-hypertrophic endometritis, moderate dilatation

may be effected under cocaine to No. 10 or 11 English bougie, and the endometrium freely touched with linimentum iodi, iodised phenol, liquor ferri perchloridi, or other agents.

Without going further into the cause of the hæmorrhage, especially if the womb were enlarged, some would now employ Apostoli's method; and though it has failed in my hands, others have been more successful. It must, however, be remembered that Apostoli's method requires many weeks to effect even a temporary *symptomatic* cure, and that it is by no means without risk, as cases published by its advocates abundantly prove. Its use also appears to be too indiscriminate.

Indeed, in reading such a valuable work as Keith's 'Treatment of Uterine Tumours by Electricity,' it is impossible to avoid feeling that it is the habitual rule to adopt Apostoli's method without any certain knowledge as to the intra-uterine condition, and in a few cases where the necessary subsequent history is given, it is clear that a preliminary exploratory dilatation would have led to a speedier and a happier result than that actually attained. At all events, it would tend much to exact knowledge if a series of cases, before being treated by Apostoli's method, could, after a previous exploratory dilatation, be tabulated according to whether the hæmorrhage was due to intra-mural, submucous, or polypoid fibroids, fungous endometritis, or malignant disease, instead of all these conditions being mixed together, as they now often are, under one common heading. Knowledge thus obtained would enable the gynæcologist to decide which cases should be treated by electricity, and which by other measures, and the current would then also be able to be applied to the exact spot where the dilatation had shown it was most needed.

I am aware that many women will undergo Apostoli's treatment who would not submit to a preliminary dilatation, but they should at all events have that precautionary measure strongly urged upon them.

Personally, however, I prefer more rapid methods than electricity in the treatment of hæmorrhage of intra-uterine origin, even though fibroids be known to be present.

Let it be assumed, then, that it is decided to dilate the uterine neck so as to admit the finger, and thus explore the cavity. One of the greatest improvements in modern gynæcology has been in the means by which this can be effected. According to Dr. More

Madden,* Philip Barrow is said to have invented the use of sponge dilators in 1539, but when Sir James Simpson revived their use, this method had been so far forgotten that he stated that "intra-uterine disease was generally considered beyond the pale of any certain means of detection or possibility of removal."

This gradual dilatation by sponge or laminaria tents, and the rapid, but still more dangerous, method of a bilateral incision of the cervix, have now been almost completely replaced by *rapid* dilatation by bougies, or other mechanical contrivances under anæsthesia. Where it used to take 24 to 48 hours to dilate the uterus sufficiently to pass the finger into its cavity, it now takes 20 to 60 minutes to dilate, and in most cases to apply the appropriate treatment.

It may be taken for granted that persistent *metrorrhagia* requires to be arrested, that an intra-uterine cause is usual, and that the shortest way to find out and remove the cause is this exploratory dilatation, the only real alternatives being removal of the appendages or hysterectomy. And removal of the appendages does not always cure the patient, for cases have been reported—as failures to cure, not as mistakes—where, after the removal of the appendages, hæmorrhage has persisted, and endometric cancer has been then suspected, or a polypus has been extruded into the vagina—as a result, according to one operator, of the absorption of the main bulk of the tumour. These were presumably cases where the cervix was out of reach, or where the hæmorrhage took the form of *menorrhagia*, and not *metrorrhagia*, and so a removable intra-uterine cause was not suspected.

Some cases have been candidly reported by the operator as mistakes, where hæmorrhage has persisted after oöphorectomy, and subsequent dilatation has revealed an intra-uterine polypus.

It is now well established, that in very many fibroid uteri the immediate cause of the hæmorrhage, even in very large tumours, such as Case 17 in the appended table, is fungous endometritis or a polypus, the removal of which would so far relieve the patient that no abdominal operation would be entertained. My surgical colleagues at the Samaritan Free Hospital are fully alive to this fact, and to them I am indebted for many cases included in the table.

Thus, in the cases under review, fourteen out of the sixteen

* 'British Medical Journal,' 1884, vol. ii, p. 1068.

fibroid uteri (88 per cent.) were found, after dilatation, to have a removable intra-uterine cause for the hæmorrhage.

I am able to show three specimens, one of which is a polypus, another cancer of the body of the uterus. I venture to affirm that neither the polypus nor the cancer could have been accurately diagnosed without dilatation, and that if they had, as an alternative, been treated by Apostoli's method or oöphorectomy, no benefit would have resulted. The third specimen is one of a submucous fibroid undergoing extrusion, with fungus endometritis all round it. I doubt whether the diagnosis in that case was possible under any known method, and its treatment would have been equally difficult. As showing the value of our present means of dilatation, I may say that all these cases died of uterine hæmorrhage some years ago, when the uterus was a sealed cavity, and their uteri were removed at the *post-mortem* examination.

When I was first attached to the Samaritan Free Hospital for Women in 1880, all suitable cases of uterine hæmorrhage not yielding to treatment had the uteri dilated by sea-tangle or sponge tents, or by incision of the cervix, and rapid dilatation was not, so far as I am aware (on the physicians' side of the hospital, at all events), adopted till the date of the first case in the table. Spite of antiseptic preparation, the use of these tents was almost always followed by pyrexia, probably sapræmic in origin, though cases of true septicæmia were not rare; and some were followed by acute pelvic inflammations, and cases of pelvic abscess, and here and there permanent ill-health, or even worse, are probably within the knowledge of most here present.

After the first tent had been in for eight to ten hours the temperature was usually approaching 100° F., with a foul vaginal discharge, and the patient was in considerable discomfort and pain.

As this first tent would rarely have dilated the internal os so as to admit the finger, two or three more had to be introduced together, and kept in for another eight hours, when the exploring finger could enter. By that time the patient's distress was often extreme, and her temperature about 101°, and possibly a rigor had occurred.

Sometimes these high temperatures were of nerve origin, as in Case 5, where the temperature rose to 107° F. within thirty minutes after the tent was introduced, and fell to 99° by next

morning when the tent was removed. In this case there was some evidence later on of chronic salpingitis and endometritis, which latter was not noticed when first dilated, owing possibly to the pressure of the tent having flattened out the rough vegetations.

Possibly if tents could be rendered with certainty aseptic, the risk of their use would not be so great; but even then, when used for the purpose of exploring the cavity, the twenty-four or thirty-six hours' continuous pain to the patient is not lightly to be passed over. The nearest approach to aseptic tents that I have seen were those prepared personally by my father; but tents must be strongly *antiseptic* as well as *aseptic* if they are to be retained in a moist cavity for so many consecutive hours with impunity. Dipping the tents in pure carbolic acid is useful, but it is in the softer pulp of the inside of the tents that the danger lurks.

During the last ten years great numbers of dilators have been recommended by their inventors, and some are shown here through the kindness of Messrs. Krolne and Sesemann. Amongst these may be named More Madden's, Lawson Tait's, Reid's, Sloan's, Dukes', Sim's, Taylor's bag, Goodell's, Coghill's, &c. These have all been invented during the time that Hegar's bougies have been before the profession, and their number and variety appear to show that there is something in Hegar's dilators which does not satisfy all the requirements desired.

London gynæcologists appear more satisfied with the bougie method, and their ingenuity has been mainly expended in modifications of the original Hegar type, such as those of Matthews Duncan, Godson, Edis, Priestley, Bantock, MacNaughton Jones, John Phillips, Hayes, Heywood Smith, and others. I have found graduated bougies the most desirable dilators, and consider Sim's three-bladed dilator a useful adjunct. The bougies mostly used are Hegar's made with longer handles than the original ones, or Leiter's, whose points and shape and length are, I think, better, as they dilate the uterus almost up to their points. The numbers are German, differing from both English and French, and represent the diameter of the bougie in millimetres.

Long-handled metallic bougies, such as those used by the late Matthews Duncan, or better still, the shape introduced by Dr. Hayes at the Royal Free Hospital, or those of Dr. John Phillips, are however much easier to use owing to the marked absence of

friction, and the longer leverage. At present I use Dr. Hayes' metallic bougies up to the diagnostic size, admitting one's little finger, No. 27 English; and then, if necessary, proceed to further dilatation, for the purpose of treating the condition found, by means of Hegar's or Leiter's bougies, No. 17 of which comes after No. 27 English.

I believe that Hegar first introduced his dilators at the International Medical Congress in 1881, but in this country they were for some years very little used. Mr. Thornton tells me he used them in private in 1882, and in November, 1883, Dr. Herman introduced their use at the London Hospital, but, except in the hands of a few such men, they were practically ignored till about 1885 or 1886; and when Dr. Lewers* read a short paper at the Harveian Society in 1887, advocating their use, the gynæcologists present opposed his views, as being not of general application, and being also dangerous.

Feeling that rapid dilatation was not taking its proper place, Dr. John Phillips† read a paper at the meeting of the British Medical Association at Birmingham in 1890, giving a table of thirty-one cases of rapid dilatation, sixteen of which were for hæmorrhage.

Since Dr. Lewer's paper, and a good deal in consequence of it, I have used this method freely, and here append a table of fifty-two consecutive cases, hospital and private, in which I have used it for hæmorrhage.

This table begins in July, 1886, at my first case of rapid dilatation, or, more exactly, at the first case in which I partially employed that method: for it will be noticed that in the first few cases, being loth to surrender tents altogether, I thought to do better by combining both methods. It was speedily evident, however, that this had no advantages, and was fraught with great risk, owing to absorption of septic products taking place more readily through the slight lacerations produced by the preliminary partial rapid dilatation. Since 1886 I have never used the slow method when the rapid was possible. A few cases, however, would still require it. Thus, Case 5 would apparently have bled to death if any delay had occurred in plugging the cervix. Another such case (No. 37) was sent to me at the hospital (July, 1891) by Mr. Edward

* 'Lancet,' 1887, vol. ii, p. 507.

† 'Lancet,' 1891, vol. i, p. 1119.

East, for extremely profuse metrorrhagia following on an abortion eight weeks before. Finding the cervix sufficiently patent to admit a Recamier's blunt curette, I incautiously introduced it, expecting to be able to detach a piece of adherent decidua. In an instant the most alarming hæmorrhage I had ever seen from the uterus occurred, and it was necessary at once to pack the uterine cavity with iodoform gauze, and to put two or three sea-tangle tents into the cervix and plug the vagina. Next day these were removed, and a piece of decidua was readily detached by the finger, and the patient remained well.

Since some form of rapid dilatation is used probably by every gynaecologist present, an apology from me is needed for the following details as to the method recommended.

After previous vaginal douches, and purgation, the patient is anæsthetised, and placed in either the lithotomy position, using Clover's crutch, or in Sim's position.

The vagina and vulva are then carefully cleansed with a 1 in 2,000 corrosive sublimate solution, and the anterior lip is seized with a vulsellum forceps, and the uterus drawn somewhat down, and held steady. This straightens the uterine curve, and prevents the great strain which must necessarily be placed upon the uterine connexions if no downwards traction is employed. Dr. John Phillips, in the paper already referred to, does not appear to consider this necessary. I cannot, however, understand how the womb can be safely dilated without.

The exact curve which the uterine cavity takes is then ascertained by a sound, as it is quite conceivable that a flabby uterus might be perforated by a bougie if pressure were exerted in a wrong direction, especially if the uterus were bound down by old adhesions in a state of stable flexion.

It is important that no bougie should be taken out till it is felt to be somewhat loosely held by the muscles at the internal os uteri, and its removal should by preference be effected by an assistant, whilst the operator holds the next size ready, warmed and oiled, for immediate insertion. The greatest resistance to rapid dilatation is offered in cases where there is a tortuous cavity with interstitial fibroids such as Case 40, though occasionally there is great rigidity from other causes. Where there is a polypus, or during and after pregnancy, the cervix yields very readily, though of the whole series of cases, Case 23 was the

most difficult to dilate, and a polypus was finally found to be present.

To curette, it is only necessary to dilate the uterus to size No. 12 Hegar, but if a certain diagnosis is required, the cervix must be dilated up to the size of one's little finger (27 English or No. 15 or 16 Hegar). No. 20 Hegar easily admits the index finger. To pass a curette, scissors, or forceps along the finger, dilatation to No. 24 Hegar is required, and to use the wire *écraseur* No. 27 or 28. A flushing curette is the best form to use, and the one here shown has a flat plate attached, to be laid on the palm of the hand, to prevent the tendency to turn round so common in most curettes.

After dilatation it is advisable to douche out the uterus, unless a flushing curette was used, and apply iodine liniment or pure carbolic acid to the lining membrane, and after carefully douching the vagina to pack it loosely with iodoform gauze. If uterine hæmorrhage continues free after curetting, as in Cases 37, 52, the uterine cavity should also be similarly packed. If it is desired to keep the uterus patent or to dilate it still further, as in Case 23, its cavity must be firmly packed with iodoform gauze or wool, after Vulliet's method.*

Rest in bed, with daily vaginal antiseptic douches, and the use of iodoform pessaries in some cases, constitute the after treatment.

Cases 10 and 31 had had their appendages previously removed, and their uteri were subsequently dilated for recurring hæmorrhage. These cases were not instances of errors of judgment, but were both due to the formation of new growths. Case 10 had had her appendages removed by Dr. Bantock six months previously for extensive disease of the tubes and ovaries, not at all for hæmorrhage, and great improvement followed the operation; but in six months she returned to the hospital, with hæmorrhage which had lasted four weeks, and when the uterus was dilated at Dr. Bantock's request, an adenomatous growth was found as stated.

In Case 31 the appendages had been quite correctly removed by Mr. Meredith for what seemed at the time of the operation to be an ordinary, though rapidly growing, intramural fibroid. For some months she kept well, and then hæmorrhage recurred at short intervals, and at Mr. Meredith's request, the uterus was dilated, and the lining membrane and the body of the uterus were

* 'Leçons de Gynécologie Opératoire.' Vulliet and Lutaud, 1890, p. 78.

found to be sarcomatous, though sufficient evidence was afforded by the microscope under Dr. Rutherford's skilful examination to show that part of the tumour was a true fibroid undergoing in its other parts sarcomatous changes.

Such a result is most disappointing after an apparent cure has resulted. Virchow, Schröder, Martin,* and Alban Doran† have all related cases where fibromyomata have degenerated into sarcomata, and the latter alludes to a case described by Dr. Finlay‡ where the tumour was distinctly encapsuled, and without doubt therefore not a primary sarcoma.

All my hospital patients were anæsthetised for me by Dr. Stormont Murray with chloroform. Dr. Murray considers that dilatation of the cervix by bougies, tightening the serre-nœud in hysterectomy, tying the pedicle in ovariectomy, sponging out Douglas' pouch, or freeing matted ovaries and tubes from their adhesions, are all similar in being liable to produce shock, intermission, or omission for two or three beats, of the pulse, and temporary cessation of respiration. Vomiting also is apt to occur during rapid dilatation. All these phenomena are lessened by deep anæsthesia, which usually allows the operation to be proceeded with. Dr. Murray, however, relates a recent case where the shock-effect was so marked that further dilatation had to be discontinued.

In my private cases ether has been given about as frequently as chloroform by Dr. Dudley Buxton, Dr. Frederic Hewitt, Dr. Murray, and others; and whilst it is certain that patients are more sick after ether, I think the dilatation itself is easier with it, for as the pelvic reflexes disappear after the palpebral reflexes it is necessary sometimes to push the anæsthetic rather deeply so as to obviate all spasm, and very deep anæsthesia cannot with safety be produced by chloroform.

Dr. Murray has observed no difference between ether and chloroform as regards the actual shock occasionally produced as the bougie expands the internal os.

In the 52 cases tabulated, the conditions found were as follows:—

* 'Centralbl. f. Gynäk.,' 1888, p. 389.

† 'Trans. Path. Soc. Lond.,' 1890.

‡ *Ibid.*, 1883, p. 177.

- 13 Placental or Membranous Retentions (3, 11, 16, 19, 25, 27, 28, 34, 37, 42, 45, 47, 52).
- Fibroids. { 5 Polypi (7, 18, 22, 23, 32).
 2 Polypi and Fungous Endometritis (6, 24).
 2 Fibroids without Fungous Endometritis (40, 50).
 7 Fibroids with Fungous Endometritis (2, 13, 14, 26, 39, 43, 48).
 8 Fungous Endometritis (1, 8, 17, 28, 33, 35, 36, 38).
 5 Granular Endometritis (20, 21, 30, 44, 49).
 5 Malignant disease (9, 10, 31, 46, 51).
 5 No *intra-uterine* cause found (4, 5, 12, 15, 41).

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The risk of rapid dilatation is extremely small. The temperature rarely rises more than half a degree, except where malignant disease is present, when a rise is apparently invariable, or where some tubal trouble exists (Cases 5, 15, 28, 29, 35, 36), in only three of which, however, and Nos. 5, 35, 36, did any pyrexia occur. In no cases has there been any serious accident or serious after-trouble. If the cervix be tough or rigid, slight lacerations of the mucous membrane usually occur, and in Cases 10, 17, 24 there were rather deep splits, all along the left side. In Case 17 the cervix was already deeply fissured bilaterally, and the tissues were very friable, and during the passage of No. 24 bougie so much pressure was put upon the anterior lip held in the vulsellum forceps, that a startling tear occurred in the line of the previous cervical tear on the left side, opening up the cellular tissue of the broad ligament, and causing free hæmorrhage from a branch of the uterine artery. This was arrested by forceps-pressure till after the uterus was curetted, when the parts were effectually united by wire sutures, and not only was there no subsequent pyrexia or pain, but there has been no recurrence of the fungous endometritis.

Such tears apparently begin at the internal os uteri, and may be suspected if a bougie pass easily after a smaller size passed with difficulty. Though no more hurry nor force should be used than is necessary, it appears that these tears, with absolute antisepsis, are not serious.

Mr. Lawson Tait's views as to the use of Hegar's dilators are peculiar.

He says "that dilatation by Hegar's dilator is just as dangerous as the use of the tangled tent, and certainly is a piece of work so arduous for the surgeons that I for one could not find time to use it. It means that the operator must sit for hours at the bedside

exercising a good deal of force, to the exhaustion of himself as well as to the infliction of much pain to the patient.”*

He evidently therefore ignores the possibility of both anæsthesia and antisepsis, and prefers a slower method by his own dilators.

No mention need be made as to the treatment required after a diagnosis of the intra-uterine condition has been ascertained; suffice it to say that the operator may be called upon to do a simple curetting, or a major operation like vaginal hysterectomy. This latter operation appears only once in the table (Case 51), and in that case, kindly sent to me by my colleague Dr. Rutherford, whose beds were then full, there was (as also in Case 30) a mucous polypus of the cervix; but as this did not appear likely to be the cause of all the hæmorrhage, the cervix was rapidly dilated, with the results described.

The proportion of cures in cases such as retained placenta, where the cause of the hæmorrhage can be at once removed, is necessarily large—practically cent. per cent.—and the same remark applies mainly in the case of polypi. When the uterus contains intramural fibroids, much depends upon whether fungous endometritis be also present as the cause of the hæmorrhage.

It is important that the uterus should be dilated right up to the fundus, as patches of hypertrophied mucous membrane are very often found high up, even at the entrance of the Fallopian tubes. Whether the fungous endometritis be general or patchy, curetting will check all hæmorrhage for many months, and if near the menopause will produce permanent amenorrhœa.

Even in cases of polypus (6 and 24) it is not uncommon to find some fungous endometritis round the pedicle or elsewhere, and the hæmorrhage will persist if this is not curetted away. I have already shown that 88 per cent. of the fibroid uteri dilated by me contained a removable intra-uterine condition which caused the hæmorrhage. Probably, therefore, a small proportion of fibroid uteri, whose appendages have been removed, were suffering from fungous endometritis. One can, therefore, hardly avoid assuming that removal of the appendages may cure fungous endometritis, but in these days of conservative surgery, one must aim at doing the greatest amount of good by the smallest degree of surgery, and I think this satisfactory minimum of surgery is to be found,

* ‘Diseases of Women, and Abdominal Surgery,’ vol. i, p. 109.

in suitable cases, in rapid dilatation and curetting, processes which restore the impaired uterine functions instead of annihilating them.

The "scrapings" in these cases of so-called fungous endometritis are mainly glandular, and appear to be hypertrophies of the lining membrane rather than any form of a true endometritis. The growth seems due to the irritation caused by the fibroid or polypus, and to the increased vascularity present. They grow more *round* the submucous fibroids, in the ruts formed by their bulgings, than on the prominences themselves, where pressure may perhaps arrest their development.

There is another form of hypertrophic endometritis, the scrapings from which are less glandular, and contain more connective tissue, and many vessels. This form occurs mainly at or after the menopause, and is therefore better called "climacteric" than "senile" endometritis. The uterus may be found to be full of a spongy gelatinous material, which can be scraped away with great ease, but which, though not malignant, is apt to recur once or twice. Cases 1, 17, 36, 38 were of this character, and Cases 20, 21, 30, 49, with a more granular mucous lining, were probably earlier stages of the same condition.

The question of intra-uterine interference when the tubes are involved remains for consideration. One naturally hesitates to dilate or curette the endometrium under these circumstances, as it is known that the mere passage of a sound without antisepsis, has set up acute tubal and peri-uterine inflammation. It is, however, proved that salpingitis is often secondary to an infective endometritis, or even, as Mr. Bland Sutton* has shown, to an ordinary endocervical adenoma. It seems sound practice therefore, where both the uterine hæmorrhage and the tubal trouble are thought to be due to an intra-uterine polypus or fungous endometritis, that a careful antiseptic exploration of the uterine cavity should be made. Cases 5, 15, 28, 29, 35, and 36 show that this may be done without much risk. In this connexion it may be observed that it is not known whether the hæmorrhage, which so often occurs in cases of salpingitis, is due to that condition or to an accompanying endometritis. Trelat,† of Paris, not only considers that an endometritis should be treated if a salpingitis exist, but is equally sure that any

* 'Surgical Diseases of Ovaries and Fallopian Tubes,' 1829, p. 265.

† 'Annales de Gynécologie et d'Obstétrique,' Paris, May, 1891.

salpingitis or other peri-uterine inflammation may be benefited or cured by curetting the accompanying endometritis. Personally I would hesitate to do this, unless hæmorrhage imperatively required checking, but many French gynæcologists agree with Trelat. On the other hand, some laparotomists would leave the case severely alone, whilst others again would disdain all intra-uterine treatment, and unhesitatingly extirpate the diseased tubes, leaving the intra-uterine condition, the actual cause of the tubal trouble, to take care of itself. This, however, is a wider subject than the one under discussion, where hæmorrhage is supposed to be the condition requiring treatment; when others with a far wider experience than my own differ so widely, I prefer not to dogmatise. Further experience will doubtless arrive at the scientific *via media*, and these cases, viewed mainly from the hæmorrhagic point of view, are meanwhile offered as a slight help towards the solution of the difficulty.

A curious class of cases remains where no intra-uterine abnormality is found, such as Cases 5, 12, &c. A history of alcoholism has been noticed in some of these cases, and also in cases where the mucous membrane has a granular feel, as above. In these pathologically negative cases the curette has sometimes been used, and sometimes not, yet improvement or cure results.

In the Lettsomian Lectures given by my father in this Society in 1864, he pointed out that one of the effects of dilatation by tents or incision was to arrest the hæmorrhage, apparently by exciting contraction of the muscles of the body of the uterus.

Possibly also the prolonged pressure of the tents would destroy any fungosities present.

Hæmorrhage and pain seem just as readily checked by *rapid* dilatation, causing contraction probably by the action of so-called uterine polarity, so ably discussed elsewhere by Drs. Champneys and Gulabin and others.

With apologies for the length of my paper, I will briefly read the conclusions which appear to me to be justified by a review of the cases upon which it is founded.

1. That where there is profuse menorrhagia, and more especially where metrorrhagia is also present, without obvious cause, the cavity of the uterus should be explored.
2. That the best way to explore the uterine cavity is to rapidly dilate the cervix with graduated bougies under anæsthesia.

No.	Date.	Name.	Age and State.	Hospital (H.), or Private (P.), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
1	July 10th, 1886	A. D.	44, M., 7 children, last 8 years ago	Samaritan Free Hospital	Almost daily loss 4 years	Uterus $3\frac{1}{2}$ ins. Reaches to umbilicus	Hegar to 10. Then sea-tangle	Papillomatous growth at fundus	Curetting	No hæmorrhage for six weeks	—
1A	Dec. 11th, 1886	A. D.	44, M., 7 children, last 8 years ago	S. F. H.	Menorrhagia last 3 periods	Uterus 4 ins. Sound causes free hæmorrhage	Hegar to 10. Then sea-tangle. Failure to dilate. Finally After	Pulpy mucous membrane throughout	Curetting	Rigor third day. Fixation of uterus. Slow recovery	No hæmorrhage for three months. Curetted again Feb. 26th, 1887, and June 5th, 1889. Reported herself well July, 1890.
2	Nov. 25th, 1886	L. C.	49, M., 1 child, last 19 years ago	P., Dr. M. B., London	Previously menorrhagia. Lately metrorrhagia and watery leucorrhœa. Pressure symptoms	Uterus $5\frac{1}{2}$ ins. Tortuous cavity	Hegar to 20 After sea-tangles for 2 days, then Hegar to 23	Sub-mucous fibroid with villous endometritis	Curetting	Hæmorrhage, Dec. 1st to 17th, 1886, and Feb. 2nd to 20th, 1887	Consulted quack in 1888, who applied chloride of zinc paste, destroying skin over abdomen and fixing tumour to parietes. Seen by Dr. Bantock, but patient declined operation. Tumour became fibro-cystic. Under Dr. Sloman of Farnham, in 1890-91. Died, Feb. 6th, 1892.
3	Nov. 4th, 1886	A. H.	36, M., last child 14 months ago	S. F. H.	Constant loss, and several floodings since parturition	Uterus 4 ins.	Hegar to 20	Placental polypus low down, near internal os	Detached with finger	No further hæmorrhage	—

No	Date.	Name.	Age and State.	Hospital (H), or Private (P), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
4	Jan. 2nd, 1887	A. B.	48, M., 1 child, 21 years ago	P.	Amenorrhœa, but constant watery blood- stained discharge	Uterus 3½ ins. Nodule at right cornu	Hegar to 18	Cavity of uterus healthy. Right tube enlarged at junction with uterus	Tubes removed by Mr. Knowsley Thornton March 1st, 1888. (Primary cancer of tube)	Rigor and pyrexia third day. Well in ten days. Discharge continues	Case described by Mr. Doran ('Trans. Path. Soc.,' vol. xxxix, p. 208), and by self in 'Trans. Obst. Soc.,' (vol. xxx, p. 194), with description of post-mortem in 'Trans. Obst. Soc.,' (vol. xxxi, p. 200). Died of pelvic can- cer, Jan. 25th, 1889.
5	Jan. 27th, 1887	M. H.	35, M., last child 12 years ago, last ab. 8 years ago	S. F. H.	Metrorrhagia 8 months. Frequent rigors 103° Fah.	Extremely blanched. Almost pulseless. Uterus bulky. No pelvic tenderness	Sea-tangle	Cavity of uterus healthy. Probably chronic salpingitis	...	30 minutes after introduction of tent, temper- ature was 107° F., fell to 102° in 90 minutes, and normal next morning when tent removed. Both tubes swelled up after rigor on Feb. 24th, 1887. Slow recovery	In Jan., 1888, some return of metror- rhagia, but otherwise well, and tubes not palpable. Was rapidly dilated, and curetted without pyrexia. Quite well, July, 1890.
6	Feb. 20th, 1887	A. T.	...	S. F. H.	Pelvic pain, and constant loss, often offensive	Uterus fixed (septic parametritis), 4 ins. Move- able body in utero	Hegar to 24	Polypus at fundus, with pulpy mucous membrane	Twisted off polypus. Curetted	Parametric exudation disappeared in three weeks.	—

No.	Date.	Name.	Age and State.	Hospital (H), or Private (P), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
7	April 20th, 1887	J. J.	39, M., 2 children, last 11 years ago, ab. 3 years ago	S. F. H.	Dyskinesia. Sanious oozing. Menorrhagia	Uterus $3\frac{1}{2}$ ins. Retroverted and prolapsed	Hegar to 27	Sessile polypus about level of internal os uteri	Enucleated	Oozing for a week. No further hæmorrhage	—
8	July 15th, 1887	J. E.	25, S.	S. F. H., Dr. H. S., South Norwood	Incessant hæmorrhage	Uterus $2\frac{3}{4}$ ins. Dextro-retro- version. Endometrium rough and vascular	Hegar to 18	Hypertrophic endometritis	Curetted	...	"No return of hæmorrhage." Feb. 16, 1888.
9	Oct. 25th, 1888	M. A.	51, M., 3 children, last 11 years ago	S. F. H., Dr. H. T. R., London	Offensive sanious dis- charge with occasional gushes of hæmorrhage	Large globular uterus, cavity $2\frac{1}{2}$ ins., very vascular	Hegar to 22	Myxo-sarcoma	Curetted	Rigor 5th day, with septic symptoms, slow recovery	Declined hysterec- tomy.
10	Nov. 5th, 1888	M. S.	36, M., 5 children, last 4 years ago	S. F. H., Dr. G. G. B., London	Diseased appendages removed May, 1888, by Dr. Bantock. Excessive loss from Oct. 1 to date	Large uterus, cervix very soft	Sea-tangle at 10 a.m. Rigor 12.30 p.m. Temp. 102° Fah., 2 p.m. Temp. 104° Fah., when sea- tangle was re- moved. Nov. 4, Temp. nor- mal. Nov. 19, Hegars to 26. Right side of uterus split	Vascular out- growth at internal os. Apparently adenomatous	Curetted and Pacquelein's cautery	No rise of temp. followed the rapid dilatation	—

No.	Date.	Name.	Age and State.	Hospital (H), or Private (P), and Medical Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
11	Mar. 6th, 1889	P., Dr. E. A. H., London	Secondary Post. Partum hæmorrhage flooding, 3 weeks	...	Hegar to 22	Small piece of adherent chorion	Blunt curette	No further loss	—
12	Mar. 28th, 1889	P., Dr. W., Brixton	...	Uterus normal size	Hegar to 16	Healthy lining membrane	Curetted	No further loss	Precisely same symptoms and treatment, Feb., 1890. Well in interval. Probably due to alcoholism.
13	May 12th, 1889	P., Dr. E. A. H., London	Metrorrhagia	Large nodular uterus	Hegar to 22	Sub-mucous fibroid with villous endometritis	Curetted	Well in 10 days	—
14	June 5th, 1889	T. F. K.	37, M., 6 children, last 5 years ago	P., Dr. E. J. B., Hoxton	Menorrhagia, 10 days' interval	Uterus reaches umbilicus, cavity 4 ins.	Hegar to 22	Fibroid at fundus. Fungous endometritis	Curetted	Relieved	Feb., 1892. Still menorrhagia every 3rd or 4th month.
15	July 8th, 1889	M. B.	34, M., 1 child, 4 months ago	S. F. H., Dr. M. B., London	Flooding since confinement at intervals	Large uterus, cavity bleeds freely. Tubes palpable	Hegar to 19	Uterus healthy. Tubes extensively diseased	...	No pyrexia followed dilatation	Appendages removed by Mr. Meredith. Matted ovaries and tubes. Double pyosalpinx. Quite well in 1891.
16	Oct. 19th, 1889	...	Aborted 3 days ago	P., Dr. H. M., Hornsey	Flooding since abortion	...	Hegar 12-20	Adherent chorion	Detached with finger	No further loss	—
17	Oct. 25th, 1889	M. C.	52, M., 10 children, last 11 years ago	P.	Polypus removed Feb., 1889. Profuse sanguinous discharge, 3 months	Uterus, very large and flabby, 5 ins. long	Hegar to 26. Cervix badly torn into cellular tissue of broad ligament. Carefully closed by wire sutures	Hypertrophic endometritis	Curetted	No pyrexia. Amenorrhœa, 3 months	Scanty menstruation, Feb. and March, 1890. Curetted July and Nov., 1890, for severe loss. Quite well, Feb., 1892.

No.	Date.	Name.	Age and State.	Hospital (H), or Private (P), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
18	Nov. 21st, 1889	J. B.	48, M., 3 children, last 20 years ago	S. F. H., Dr. F. M.,	Flooding 3 years ago. Menorrhagia ever since	Fibroid polypus can just be felt	Hegar to 27	Egg-sized polypus growing from anterior wall of uterus	Wire écraseur	Temp. 100° 2nd day No further loss	—
19	Jan. 3rd, 1890	P., Dr. C. C. B., Norwood	Hæmorrhage and suppræmia (103° F.) since confinement 5 days ago Profuse menorrhagia, lasting 14 days, for 18 months	... Large soft uterus 3 ins. Lining membrane rough and vascular Uterus globular but only 2½ ins. long	Hegar 10-20	Piece of retained membrane, partly adherent at fundus	Blunt Curette	Hæmorrhage and pyrexia both ceased ...	—
20	Feb. 10th, 1890	S. D.	46, M., 6 children, last 5 years ago	S. F. H.	Profuse menorrhagia, 3 years	Uterus 4 ins. long	Hegar to 18	Granular endometritis	Curette	No loss in hospital	Well in Oct., 1890. Amenorrhœa, 2 months.
21	Feb. 24th, 1890	M. A. C.	49, W., 3 children, last 22 years ago	S. F. H.	Profuse menorrhagia, 3 years	Uterus 4 ins. long	Hegar to 16	Polypus at fundus	Curette	... Quite well, Christmas, 1891.	—
22	July 15th, 1890	G. J. P.	52, M., no children	P.	Constant hæmorrhage, 6 months	Uterus 4 ins. long	Hegar to 27	Fibroid polypus, broadly attached at fundus	Wire écraseur	Some oozing for 10 days	—
23	Oct. 6th, 1890	S. B.	42, S.	S. F. H.	Polypus removed 3 years ago. Lately recurrence of hæmorrhage	Uterus 4 ins. long	Hegar to 15, but rigidity of interior os prevents further dilatation, so sea-tangle inserted Oct. 9, and further dilated with iodoform tampons		Enucleation		

No.	Date.	Name.	Age and State.	Hospital (H.), or Private (P.), and previous Medical Attendant.	Symptoms	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
24	Oct. 25th, 1890	Mrs. D.	33, M., 2 children, last 5 years ago; molar pregnancy, March, 1889	P., Dr. J. B. H., West Norwood	Menorrhagia, 5 years. Pruritus pudendi	Uterus very low, 3½ ins. Kraurosis vulvæ	Hegar to 20. Uterus gave way laterally as No. 16 bougie was passing	Soft flaccid polypus, size of walnut at orifice of left Fallopian tube. Some granular endometritis Retained adherent decidua	Twisted off polypus and curette	Periods con- tinued excessive till Feb., 1891, when they became quite normal. Uterus 3 ins.	"Quite well," Christ- mas, 1891.
25	Nov. 10th, 1890	P., Dr. H. C. C. S., Hampstead	Hæmorrhage after abortion (2 days) Constant loss of blood, 3 years	...	Hegar 12-18	Retained adherent decidua	Detached with finger	No further loss	—
26	Nov. 24th, 1890	A. W.	50, M., No children. No abortion	P., W., Dr. J. W., Crandall	Uterus 3½ ins. Cervix soft	Uterus 3½ ins. Cervix soft	Hegar to 26	Fibroid polypus posterior wall. Fungous endometritis round pedicle	Scissors curette	...	In January, 1891, had a profuse menstrual loss. Then amenor- rhea to August. Three losses between Aug. and Dec., 1891. Severe loss, March, 1892. Pro- bably more endo- metritis.
27	Jan. 8th, 1891	P., Dr. C. G. B., Norwood	Secondary post partum hæmorrhage	...	Hegar to 27	Small nodule of placenta, very tightly adherent	Detached by fingers	No further loss	—
28	Jan. 13th, 1891	L. M.	28, S.	S. F. H.	Metrorrhagia, 5 months	Uterus 3½ ins. Left tube palpable	Hegar to 18	Fungous endometritis	Curette	Tube more swollen, but no pyrexia	Ante-menstrual dys- menorrhœa, but no metrorrhagia, Feb., 1892.

No.	Date.	Name.	Age and State.	Hospital (H.), or Private (P), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
29	Feb. 9th, 1891	E. T.	..	S. F. H., Dr. B., Bury St. Edmunds	Profuse menorrhagia since July, 1890	Uterus 4 ins. Some old left tubal swelling	Hegar to 26	Small flaccid polypus at fundus	Twisted off	No pyrexia. Temporary relief from hæmorrhage	Profuse period Feb., 1892, followed by left-sided pain. Going to St. Mary's Hospital, Manchester.
30	April 3rd, 1891	F. J. A.	53, M., 3 children, last 22 years ago	P., Dr. T. J. B., Crondall	Flow of bright blood on rising, and at defæcation since Oct., 1889	Four or five mucous polypi hanging from endocervix. These were twisted off, but sound caused free hæmorrhage from uterine cavity, which was 3 ins. long	Hegar to 16	Very vascular granular lining	Curetted	...	Quite well, Christmas, 1891.
31	April 20th, 1891	J. A. W.	48, S.	S. F. H., Mr. W. A. M., London	Metrorrhagia 4 years ago. Appendages removed by Mr. Meredith, March, 1889. Well till Christmas, 1889, then slight metrorrhagia. Now daily loss, often profuse	Uterus 3½ ins. Mobile. Lining membrane very rough and vascular. Uterus bulges to left, and there is thickening of left broad ligament. Vagina greatly contracted	Hegar to 22, with difficulty owing to atrophied vagina	Sarcomatous growth, growing from fundus and left side	Partial curetting, but parts extremely friable. Hysterectomy useless	Temperature rose to 103° next day, and was normal 3rd day	July, 1891. Rapid recurrence. Growth protruding through exterior os uteri, with signs of septicæmia. Redilated, and part removed. Temperature rose next day to 104°-8. Death, Nov., 1891.

No.	Date.	Name.	Age and State.	Hospital (H), or Private (P), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
32	May 4th, 1891	P. S.	42, M., 2 ab., 11 years ago	S. F. H., Mr. A. D., London	Menorrhagia and dirty water discharge for 18 months	Uterus $3\frac{1}{2}$ ins. Cervix very soft	Hegar to 18	Mucous polypus growing from level of interior os upward into cavity	Twisted off	Amenorrhœa to July	Quite well, Mar., 1892.
33	May 5th, 1891	W. W.	24, M., 1 child, 6 months ago	P.	Almost constant loss since March 5, with left ovarian pain	Uterus $3\frac{1}{2}$ ins.	Hegar to 18	Left side of uterus rough and vascular	Curetted	...	Quite well, Jan., 1892.
34	June 15th, 1891	M. H.	38, M., 12 children, last 12 months ago. 1 ab., 10 months ago	S. F. H., Dr. J. M. R., Colchester	Secondary P.P.H. 8 weeks after abortion, and now almost daily loss	Uterus 4 ins., very capacious cavity	Hegar to 22	Pulpy redundant decidua at fundus	Curetted	No further loss	—
35	June 20th, 1891	...	33, M., 1 child, 10 years ago	P., Dr. C. P. G., Camberwell	Was curetted June, 1890, for metrorrhagia, then amenorrhœa for 4 months, and now as bad loss as before	Acute endometritis. Sound causing agony. Tender body in Douglas Pouch. Probably tube	Hegar to 16	Acute endometritis	Curetted	Some fresh swelling of left Fallopian tube followed, temperature 100° for 3 days. Catamenia regular but scanty till October	Amenorrhœa, Oct., 1891, to Feb., 1892. Tube still swollen and tender.
36	July 6th, 1891	M. M.	44, M., 2 children, last 20 years ago	S. F. H.	Hæmorrhage or offensive watery discharge, 4 years	Uterus 4 ins. Pulpy shreds come away in discharge. Right tube swollen.	Hegar to 20	Fungous endometritis, confined to right cornu (?) involving tube	Curetted	Temperature rose to 100°·4, 2nd day with sore throat. No further loss	Amenorrhœa, July, 1891, to Feb., 1892.

No.	Date.	Name.	Age and State.	Hospital (H.), or Private (P.), and previous Medical Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
37	July 9th, 1891	E. A. P.	27, M., 1 child, 1 year ago, 1 ab., 2 months ago	S. F. H., Dr. E. E., London	Loss since abortion, with several severe floodings	Uterus $4\frac{1}{2}$ ins. long still admits 18 English bougie. Blunt curette used. Sudden terrible hæmorrhage, almost to syncope	No anæsthesia being available, uterus was packed with iodoform gauge, sea-tangle introduced into cervix, vagina plugged Hegar to 20	Next day could introduce little finger, and found partially detached piece of decidua at right cornu	Blunt curette	...	No further loss, Feb. 10th, 1892.
38	July 13th, 1891	E. H.	51, M., 3 children	S. F. H., Dr. S. S. L., New Cross	Menorrhagia 4 years, constant hæmorrhage 4 months, Cured by Dr. Matthews	Uterus $3\frac{3}{4}$ ins.	Hegar to 18	Senile fungous endometritis	Curette	Watery oozing 3 days	March, 1892. More hæmorrhage. To be curetted again.
39	Oct. 3rd, 1891	T. C.	40, M., 2 children, last 8 years ago	P., Dr. T. H. G., London	18 months ago, had profuse menorrhagia 3 months, with nausea Menorrhagia 9 months, occasional retention of urine	Uterus bulky, $3\frac{1}{4}$ ins. Bleeds freely	Hegar to 18	Mucous membrane pulpy, and small fibroid (intramural) at fundus	Curette	Next "period" natural. Somewhat profuse in Dec., 1891	Quite well, Mar., 1892.
40	Oct. 5th, 1891	S. P.	40, S.	S. F. H., Dr. T. B. F., Mirkfield	Menorrhagia occasional retention of urine	Impacted retro-uterine fibroid polypus (?) bulging through cervix	Hegar to 15. Cervix very rigid	All fibroids intramural. Healthy lining membrane	Pelvic fibroid raised into abdomen by hydrostatic pressure	Temperature rose to $100^{\circ}\cdot6$ day after reduction of fibroid	No menorrhagia or bladder trouble, Feb., 1892.

No.	Date.	Name.	Age and State.	Hospital (H), or Private (P), and previous Medical Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
41	Oct. 5th, 1891	M. E. A.	38, M., 1 child, 15 years ago, 3 ab., last 2 years ago	S. F. H., Dr. T., Brondesbury	Slight metrorrhagia, 12 months, profuse 10 weeks	Uterus $3\frac{1}{4}$ ins. retroverted and low	Hegar to 17	Healthy	Reduced uterus and fitted hodge	...	Quite regular, March, 1892.
42	Oct. 7th, 1891	P., Dr. W. J. C. S., London	Hæmorrhage after abortion	...	Hegar 10-16	Adherent patch of decidua	Blunt curette	No further loss	—
43	Oct. 12th, 1891	C. F. H.	40, S.	S. F. H.	Menorrhagia 10 months (10-14 days)	Large fibroid to navel, uterus 4 ins.	Hegar to 20	Intramural fibroid. Mucous membrane granular	Curetted	Watery oozing for a week	Period only lasts 4 days, Feb., 1891.
44	Oct. 18th, 1891	M. E. P.	30, M., 2 children, last 4 months ago 3 ab. prior	P.	Metrorrhagia since parturition	Uterus subinvo- luted and retroverted, with adhesions	Hegar to 21	Mucous mem- brane granular	Curetted uterus, and broke down retro- uterine adhesion. Introduced hodge	...	Writes from Havre Feb. 20th, 1892:— "Loss much less for 3 months, last month rather more."
45	Nov. 17th, 1891	D. D.	26, M., 1 ab., 5 years ago, now pregnant	P., Mr. S. B., London	Pregnant $3\frac{1}{4}$ months. Daily hæmorrhage	Molar pregnancy suspected, so ergot given. Hydatid mole expelled. Severe hæmorrhage 6th and 7th days	Hegar to 22	A few cystic bodies found imbedded	Curette	No further loss	Quite well, Mar., 1892.

No.	Date.	Name.	Age and State.	Hospital (H.), or Private (P.), and previous Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
46	Nov. 23rd, 1891	P. E. M.	60, S.	S. F. H., Dr. L. L., Bexley Heath	Hæmorrhage off and on last 4 years. Very profuse since July, 1890, with severe leucorrhœa. Uterine colic Fleshy mole came away yesterday, recurring post-partum hæmorrhage, which became alarming next day	Uterus not duly mobile. Bulky. Appears to contain growth	Hegar to 20	Myxo-sarcoma of fundus and back of uterus, surrounding parts involved	Curetted deeply into uterine tissue	Severe pain, and temperature rose to 100° F.	March, 1892 :—"Freer from pain and hæmorrhage."
47	Dec. 11th, 1891	...	24, M., 1 ab. day prior	P., Dr. E. T. G., Fulham	Fleshy mole came away yesterday, recurring post-partum hæmorrhage, which became alarming next day Excessive menorrhagia, 7 days' interval	Shreds of decidua had been already removed, but hæmorrhage did not cease	Dilated to 24	Very small piece of membrane adherent. Intramural fibroid anteriorly	Blunt curette	No further loss	Quite well, Mar., 1892.
48	Jan. 5th, 1892	...	S.	P., Dr. F. H., Hampstead	Loss for 3 weeks, and then interval of 16 days, and now constant loss for 8 weeks	Uterus enlarged	Dilated to 22	Fibroid size of cocoa-nut in anterior wall. Much fungous endometritis all round	Curette	Next period much less	March period somewhat profuse.
49	Jan. 12th, 1892	...	41, M., 4 children, last 6 years ago, 3 abortions prior	P., Dr. A. L., Walworth	Constant daily hæmorrhage, 5 months	Uterus 8½ ins. Fibroid	Uterus already dilated with sea-tangles by Dr. Langley	Roughened mucous membrane	Curette	No further loss	Quite well, Mar., 1892.
50	Feb. 15th, 1892	J. F. V.	28, S.	S. F. H.			Hegar to 16	Large anterior intramural fibroid. Lining healthy	...	No loss whilst in hospital 14 days	—

No.	Date.	Name.	Age and State.	Hospital (H), or Private (P), and previous Medical Attendant.	Symptoms.	Physical Signs.	Method and extent of Dilatation.	Condition found in Utero.	Treatment adopted.	Immediate Result.	Subsequent History.
51	Feb. 15th, 1892	M. A. D.	63, W., no children	S. F. H., Dr. H. I. R.	Watery discharge 9 months. Hæmorrhage 3 months	Uterus $2\frac{1}{2}$ ins. Retroverted. Mobile. Vagina small	Hegar to 18	Cancer of fundus and right side of body	Feb. 16, temp. 100° F. Feb. 22, vaginal hysterectomy Curette	Vaginal wound closed 6th day. Acute mania 9th to 13th day	Died suddenly, when apparently convalescent, 16th day.
52	Feb. 29th, 1892	E. J.	28, M., 4 children, 2 ab. Last 6 weeks ago	S. F. H., Dr. H. T. R.	Constant hæmorrhage since abortion; very severe last 14 days	Uterus $3\frac{3}{4}$. Large, flabby, and antverted	Hegar to 18	Pulpy decidua at right cornu		Hæmorrhage free, so plugged uterus with iodoform gauze after curetting. No further loss.	—

3. That with rigid antisepsis there is practically no risk, and very rarely any subsequent pyrexia, unless malignant disease or salpingitis is present.

4. That even where tubal disease is present or suspected, exploratory dilatation of the cervix, *for metrorrhagia of apparently intra-uterine origin*, is not necessarily contra-indicated, salpingitis being often secondary to and aggravated by intra-uterine disease. Here again antisepsis is all important.

5. That where fibroids of the uterus are evidently present, the immediate cause of the hæmorrhage may be a removable one, such as a co-existing polypus or a fungous endometritis, and that therefore, the uterine cavity should be, when practicable, explored, before removal of the appendages or hysterectomy is entertained.

6. That in some cases dilatation alone suffices to greatly relieve both the hæmorrhage and pain.

7. That if an exploratory dilatation were more often adopted prior to the employment of Apostoli's treatment, it would tend to a more exact knowledge of its applicability, and put its use on a more scientific basis.

Mr. ALBAN DORAN said that rapid dilatation as a means of diagnosis was particularly valuable, since our knowledge of intra-uterine disease was not so satisfactory as our acquaintance with those very marked affections to which the ovaries and tubes were subject. The precise nature of endometritis remained uncertain; indeed, authorities were not agreed as to the normal changes which the endometrium underwent during each menstrual cycle. Yet many of the graver inflammatory diseases of the appendages took their rise from the lower part of the genital tract. When we knew more about endometritis we should be the better able to cure it, and thus to save tubes and ovaries, or even to spare the endometrium from the curette. Dilatation would help us to detect early disease and to nip it in the bud.

Mr. KNOWSLEY THORNTON agreed with the first and second of the author's conclusions, and he was also sure of the value of antisepsis. He never passed a sound but he followed it with a probe, on which was twisted a bit of cotton-wool dipped in iodine, and he left this in the uterus for a few seconds; he had not found pyrexia follow dilatation, even in malignant cases. He did not see why antiseptics were all-important in pyo-salpinx cases, because in those the woman could not be protected from sepsis. In many cases of uterine fibroid it was quite impossible to explore and to render the uterine cavity aseptic; it was far better to proceed with the major operation at once. He had found hæmorrhage and pain relieved by operation, sometimes in a way not easy to explain. He thought that a debate on this subject would prove useful to the profession at large, for there was still much ignorance on this subject, and he quoted, in conclusion, a case of fungous endometritis in which, for want of dilatation, carcinomia of the uterus had been diagnosed. Hysterectomy

had been proposed, and, but for an independent opinion, would probably have been carried out.

Dr. HAYES agreed with the author's general conclusions, which accorded with his own experience. Long ago he came to the conclusion that dilatation by tents was painful and even dangerous, because it led to cellulitis and sepsis, and he had therefore adopted Hegar's dilators, which he had made with long handles and a more rounded extremity. He had found fixation of the uterus by vulsellum unnecessary, for where it was done it incommoded the passage of the dilator into the uterus. Before removing tumours he preferred, after rapid dilatation, for diagnosis, to use laminaria tents, as under the influence of the latter the uterus became softer and paralysed. The only case in which he had met with accident in rapid dilatation was the following :—A woman past the climacteric was suffering from profuse and watery discharge. As he dilated, the uterus ruptured, both at the side and at the fundus, although no force was used. At the *post-mortem* examination there was found fatty degeneration of the liver and of the heart; the uterus itself was very soft, and apparently fatty also. He would hesitate to dilate if acute salpingitis were present.

Dr. WILLIAM DUNCAN thought that in all cases rapid dilatation should be resorted to in preference to gradual, there being far greater risk with the latter. The only exception he would make would be in the case of malignant disease of the fundus. He held that the uterus should be fixed with a vulsellum, as a case had been recorded in which the organ had been torn from its connections where dilators alone had been used. Where the cervix could not be easily gripped he usually made a small bilateral slit, and then passed one blade inside the cervix and one out. In fibroids the operation of hysterectomy or of removal of the appendages should be preceded by dilatation of the cervix. He related two cases in which, having neglected to previously dilate the cervix, he removed the appendages for fibroid, without checking the bleeding. One of the patients subsequently returned. He then on dilatation found a pedunculated fibroid as large as an orange, which he removed with the écraseur, with the result that the hæmorrhage almost entirely stopped, though some intramural fibroids still remained. Slight lacerations of the cervix were of no moment and healed up rapidly; they were obviated in the virgin uterus by lateral incision. He had found that dilatation could be carried out without the aid of an anæsthetic in many cases in multiparæ. He agreed cordially in the advantage of thorough asepsis. After dilating he introduced iodised phenol and placed an iodoform bougie in the canal, which was kept in place with a plug of glycerine.

Dr. JOHN WILLIAMS did not know why Hegar's dilators were considered to be so good, and why tents were so depreciated. The latter had, till comparatively lately, been constantly used with very few bad results, if the patients were kept in bed and on their backs. Rapid dilatation by bougies was better when pregnancy had recently occurred, but in women who had not been pregnant for a long time the risk attaching to their use was very great. To dilate a uterus by means of them so that the forefinger could be introduced as far as the fundus meant that the cervix must be lacerated in every case, and the tear began at the internal orifice and extended downwards; therefore he could not see why incising the external os should stop it. Such a laceration would leave a scar which might give rise to trouble afterwards. It was not necessary to dilate a uterus for diagnosis so that a forefinger could be passed into it—endometritis, for instance, could be easily told by the way in which a sound

passed gently over the surface of the mucous membrane produced hæmorrhage. For the treatment of that condition rapid dilatation was most valuable, the canal being dilated up to the size of a No. 18 (English), and the curette then being freely used. In cases of hæmorrhage the cavity of the uterus should not be explored until the case had failed to be controlled by medicine. The risk of rapid dilatation was slight, but in one case, about six days afterwards what appeared to be a hæmatocele developed, but the patient did quite well. He had dilated safely in instances in which there were lateral swellings on each side of the uterus. If hæmorrhage were present in association with tubal disease, its occurrence was probably accidental. He admitted the advantage of dilating in many cases of fibroids; at any rate, if this were done more frequently a large number of women might have their ovaries saved.

Dr. HEYWOOD SMITH said that though he did not hear the first part of the paper yet he would like to make a few practical remarks: and first, as to what had been said about the cervix being held by an assistant, he quite disagreed with that method; he considered it far better that it should be held by the operator himself, as then he could tell exactly the force he was using, and feel more correctly the onward progress of the dilator. Then, in using the ordinary spoon, or loop-shaped curette, the scraping was made in lines, and so some of the surface might easily be passed over, whereas if the "dredge curette" of Dr. Robert Bell was used it would be found to scrape all the lining of the uterus, and it brought away all the *débris* entangled in its wires. He also considered that it was better to plug with a long strip of gauze rather than with wool, as the wool was more difficult of extraction, and some of it might even be left behind. He thought each dilator should have a notch marking the normal length of the uterus for the better guidance of the operator. He quite agreed with what had been said by Dr. John Williams as to its not being necessary to dilate more than to about Nos. 15 to 18 (English) in order to make ordinary applications to the interior of the uterus. He did not see any reason why dilatation should not be had recourse to after the menopause; in fact, many of the cases that required dilatation were those in which there was a suspicion of malignant disease occurring late in life. Nor did he consider tubal disease a bar to dilatation. He had recently a case in his hospital where a pyo-salpinx had emptied itself during menstruation, and he thought it not improbable that dilatation and perhaps curetting might lead to the evacuation of the contents of the oviduct.

Dr. PETER HORROCKS said that all were agreed as to the advisability in certain cases of dilating the uterus for the purposes of diagnosis and treatment. The question was what were the best means of accomplishing this purpose. He maintained that any particular means might be the best in some cases, and not in others. He considered Hegar's dilators, and the various modifications of them, of very great utility, and that they saved time, trouble, and expense, besides lessening danger in many instances. But he demurred to the assertion that they or any other form of rapid dilatation was the best in all cases. He considered that, in addition to the instances mentioned by Dr. John Williams, with whose statements he entirely agreed, it was very undesirable to attempt the rapid dilatation of the uterus after the climacteric, because it had lost much of its elasticity, and had become less muscular and more fibrous, and consequently more rigid. When it was necessary to dilate up such a uterus, he preferred a more gradual method. Moreover, experience in the use of rapid dilators taught one that in some cases it would be risky to per-

severe. Each successive dilator required so much more force that obviously the parts would not yield, and to go on would result in laceration. He believed that when a uterus, not having been recently pregnant, was dilated so far as to enable a digital exploration to be made, there was always more or less laceration of the mucous membrane, at all events. They generally began at the internal os, and spread downwards. Occasionally they split deeply into the cellular tissue of the broad ligaments. These lacerations did not usually cause any immediate serious consequences. He mentioned a case in which a Hegar's dilator was passed right through the uterine wall. No untoward result followed. The patient was the subject of some lung complaint, from which she died four months afterwards, and at the *post-mortem* examination the scar caused by the dilator was distinctly visible. He said that the methods of dilatation were two, namely, mechanical simply, as by Hegar's dilators, and partly mechanical and partly physiological, as by tents and other gradual methods; because in these the dilatation of the cervix caused a contraction of the body and fundus of the uterus, and this produced a corresponding relaxation of the cervix by the law of polarity, thus facilitating dilatation.

Dr. LEWERS said that reference had been made to his paper on "Rapid Dilatation of the Cervix," read before the Harveian Society, some five years ago. Many then present who were opposed to the method had since become strong supporters of it. He himself now considered that rapid dilatation was not a method to be universally applied in all cases, but that it was of special value where dilatation was required at no long time after a confinement or miscarriage, or in cases of molar pregnancy. For such cases it was a perfect method. On the other hand, in cases altogether apart from pregnancy, or in those who had never been pregnant, rapid dilatation often meant laceration, and for such cases dilatation by means of laminaria tents, rendered aseptic and antiseptic by prolonged immersion in a 1 per cent. solution of corrosive sublimate in absolute alcohol, was the best method.

Dr. CHAMPNEYS said that, at so late an hour, he would only touch on two points, the question of antiseptics and the physiology of the dilatation of the cervix. As regarded the question of antiseptics, it had been too easily taken for granted that Hegar's dilators were essentially aseptic, and laminaria tents essentially septic. Now, either dilators or tents, unless carefully prepared, would introduce germs from without. The cervico-uterine canal could not be taken as necessarily aseptic, nor could it be rendered easily aseptic, but the strong antiseptics which could be used for tents or dilators (the best being glycerine of corrosive sublimate, 1 in 1,000) would help to render it aseptic. There remained the question of the changes in a tent left *in situ* during many hours. He thought, however, that with care no cause for apprehension need exist if the vagina were packed with a good antiseptic, such as absorbent gauze, soaked in corrosive sublimate solution. He knew that such a plug remained sweet for days. He believed that it was possible to be as aseptic with tents as with dilators. The second point was the physiology of the cervix, which had not been alluded to. It must be remembered that dilatation of the cervix was a physiological act. The cervix would not be forced, but it could be induced to yield. When it yielded, it also freely secreted. This connection of yielding and secretion was familiar in the process of labour, but it could also be seen in the non-pregnant condition. When the cervix was moist, it was dilatable. When it was dry, it was rigid, and would tear rather than yield. In this latter condition, any attempt at rapid

dilatation was generally a failure, and was even dangerous. The best thing to do was to attempt to get the cervix softened, and that was best effected by the gradual influence of a tent. In such cases his method was to disinfect the vagina thoroughly, then to pass the smaller sizes of Hegar's dilators till he found what sized tent could be accommodated without stretching, to insert the tent, to pack the vagina antiseptically, and then, after some hours, to complete the dilatation rapidly. The tent had often softened the cervix most effectually, and, in any case, gave very valuable information, by the presence or absence of constriction, as to the quality of the cervix and the size of the canal. In cases of imperfect abortion, where the softening was already present, rapid dilatation alone generally sufficed.

Dr. AMAND ROUTH, in reply, thanked the Fellows for their kind criticisms, and for the information afforded in the discussion. He was always glad to have Mr. Doran's pathological support. Mr. Thornton's view that it was not only dangerous, but often useless, to dilate in cases of large fibroid, was somewhat disproved by Case 17, where the uterus was originally 8 inches long, its size being almost entirely due to a large fibroid polypus, which was removed, fungous endometritis subsequently occurring. He did not agree with Dr. Duncan, that tents should by preference be used in fundal cancer, first because no one could diagnose the cancer with certainty before dilatation, and secondly, because septic changes in malignant growths, with foul discharges, were very apt to occur, and cause septicæmia. Dr. Duncan's case of hæmorrhage persisting after removal of the appendages, and the subsequent discovery of a polypus after dilatation, was a strong argument as to the value of a *preliminary* exploratory dilatation. He had not met with cases which could be dilated without anæsthesia except *post partum*. He regarded Dr. John Williams' and Dr. Champney's views as to the possibility of antiseptics being carried out equally successfully with tents or Hegar's dilators as most valuable, and they would cause him to try tents again in the cases advised. The length of time during which tents had to remain *in situ*, and the impossibility of making the endocervix really aseptic beforehand, must not be overlooked. He rarely went beyond No. 15 (Hegar), admitting the little finger for diagnostic purposes and curetting, but he did not agree with Dr. Hayes, that it was safe to reach this degree of dilatation in 10 minutes. By holding the vulsellum forceps vertically instead of horizontally, dilatation was not interfered with. If much resistance was offered to the passage of a bougie, he held the vulsellum forceps himself, otherwise the fingers of his second hand remained in the vagina, to guide the bougie into the cervix. He thought Bell's curette useful to curette low down in a symmetrical uterus, but useless in cases of bulging fibroids, or where the fundal zone required curetting. He had found tamponning after Vulliet's method very useful sometimes. He did not share Dr. Horrocks' objection to dilate after the climacteric, believing that a certain differential diagnosis between cancer and fungous endometritis could only thus be made. He was sorry to find that Dr. Lewers was not so enthusiastically in favour of rapid dilatation as he used to be, but he accounted for it by observing that Dr. Lewers used short Hegars, instead of the long frictionless metallic bougies. He thought the remarks of Dr. Champneys as to the physiological process of dilatation very valuable, and likely to lead to a better selection of cases in the future.

April 11th, 1892.

FURTHER CASES ILLUSTRATIVE OF HEPATIC SURGERY.

By J. KNOWSLEY THORNTON, M.B., M.C.

I HAVE on three former occasions made communications to this Society on hepatic surgery, and I trust that I shall not weary you by returning to the same subject, for it seems to me to the advantage of those interested in a subject to have all that relates to it published in the same publication or series of publications. Thus, contributions to other branches of surgery have been made entirely to sister societies, or published always in the same medical journal, and are thus easy of reference.

The first case I have to record on the present occasion is that of a widow lady of about 40 years of age, mother of one daughter. She had suffered severely during many years with attacks of biliary colic, and had been frequently to Carlsbad without obtaining any permanent relief. I saw her in consultation with my friend Dr. Philpot, and we decided that cholecystotomy should be performed. Just before I operated she had undergone a long course of yellow soap treatment, about which she was very caustic, comparing its discomforts very unfavourably with the results of surgery. I operated on April 18, 1891, and removed four large angular stones and a quantity of small ones from the gall-bladder, and then found that another large angular stone was impacted in the cystic duct, and that the duct had contracted firmly behind it. I needled it carefully into several fragments through the duct wall, then crushed the fragments by finger and thumb pressure, and by pressure with nasal polypus forceps with their blades guarded with india-rubber tubing, and left the *débris* in the duct, with every confidence that it would soon find its way down into the common duct and on into the intestine. The gall-bladder was sutured into the upper part of the abdominal incision and drained. The patient made an excellent recovery without any untoward symptom.

I may mention here that inquiries as to the present health of the patients I have previously operated upon, all of whose cases have been reported to this Society, have elicited most satisfactory reports. There do not seem to be any after troubles or any liability to recurrence. A word as to the method of suture in fixing the gall-bladder into the abdominal incision. I have tried various plans, and I have come to the conclusion that the best plan is to

first suture the upper angle of the wound in the gall-bladder into the upper angle of the peritoneal wound (gall-bladder to peritoneum merely), to keep on the needle and tie the suture, leaving a long end, to make a continuous suture of the left edge of gall-bladder to peritoneum till the lower angle of the wound in the gall-bladder is reached, then to take off the needle and thread it on to the end left loose at the first tie, and make a continuous suture down the right side of the wound in the gall-bladder, then tie the two ends of the suture together where they meet in the lower angle of the gall-bladder wound, using fine silk (No. 1 twist), and leaving the suture with short cut knot to take care of itself or be taken care of by the peritoneum in the deep recesses of the wound. I find that with this method of suture the wound in the gall-bladder closes more quickly than with interrupted points, and there is just enough of the bag mouth principle in it to make the opening fit pretty closely round a small rubber tube. If the suture, on the other hand, is made quite continuous all round, it is apt to pucker up a little too much, and, pressing upon the tube, gives pain, and it is not quite so easy to bring the peritoneum closely together with the first abdominal wall suture below the gall-bladder. This first suture should be applied as closely as possible to the point where the lower angle of the gall-bladder is, then when it is tied there is only a very small slit of the parietal wound left open for the protrusion of the tube from the gall-bladder.

When should the tube be removed from the gall-bladder? is a question I am often asked. Well, experience here again has taught me to use a very much smaller tube than I did at first, and to leave it in a much shorter time. I think it is well in most cases to leave it in for a week at least, so that the tissues of the abdominal parietes round it, from the opening in the gall-bladder to the skin opening, may grow well together, and form a firm channel round it; then it may usually be removed with advantage, more of the contents of the gall-bladder finding their way through the ducts, and less into the dressings at once. If it be too soon removed, on the other hand, the bile and mucus from the gall-bladder flow down between the edges of the parietal wound and make it heal badly, or even open up in its whole extent, an obviously undesirable accident, and one which makes the after healing of the incision a long and troublesome business, with careful

daily strapping and an amount of personal care which few busy surgeons can give daily.

CASE 2.—This is a very interesting one. The patient, a married lady, was sent to me by Dr. Wilson, of Oxford. She had a long history of attacks of biliary colic; they had recently become very severe, and there had been almost continuous pain through to the back, requiring a good deal of morphia. She had emaciated and was somewhat jaundiced. On making an examination of the abdomen, I found what I had no doubt was a large barrel-shaped stone in the gall-bladder, and below this on deep pressure I thought I could outline another similar stone about the junction of the cystic and common ducts. Operation was decided upon, and was performed on April 27, 1891. On exposing the gall-bladder, I found its wall full of nodules of malignant growth, and similar growth disseminated through the liver round about the gall-bladder in such a way that there could be little doubt that it was secondary to the disease in the gall-bladder. I found, as I had diagnosed, these two barrel-shaped stones, one in the upper part of the gall-bladder, one partly in the gall-bladder and partly in the cystic duct, but not so low as I had expected to find it. The jaundice was not due, as I had thought, to this stone projecting into the common duct, but to the malignant disease obstructing the common duct. I had difficulty in finding a sufficiently healthy spot in the gall-bladder to make my incision through. I found the ducts below the stone healthy, and I determined to entirely close the wound in the gall-bladder and drop it into the peritoneum. I was led to this decision from the fear, I may say certainty, that if I sutured it into the abdominal incision the malignant growth would spread into its tissues, and we should have a biliary fistula with malignant edges, a misery so great that I felt fully justified in running the undoubtedly considerable risk of complete suture and dropping of the gall-bladder, suture in unhealthy tissue, and possibility of distension of the gall-bladder by obstruction to the ducts from the malignant deposits around them. Happily the wisdom of my procedure was justified by the result. The abdominal wound healed well, and the case was in all respects a smooth one but for the excessive pain in the back, which continued, and was evidently due to the progress of the disease in the liver. The other pain and discomfort she had suffered quite disappeared.

One can readily understand that tissues infiltrated with nodules of cancer pressing constantly on these hard stones must have caused much suffering, and their removal proportionate relief. I did not hear from Dr. Wilson after her return home, but I heard by chance recently that she only lived a few weeks.

I think there can be little if any doubt that the long-continued irritation caused by the stones had produced the malignant disease, and that an earlier operation might have saved a valuable life. I do not believe that it would have been possible before opening the abdomen to diagnose the malignant disease as coincident with the stones, and I think the operation was fully

justified by the successful removal of the stones, and by the partial relief thus afforded.

The satisfactory healing of the wound in the diseased gall-bladder is a very interesting fact, both for the pathologist and the surgeon; some of my sutures were almost in the malignant nodules. I used a row of fine silk interrupted sutures, and then a continuous suture of the same silk applied widely in the peritoneal covering of the gall-bladder, so that when it was tightened the first row of sutures was practically buried.

The success attending this method of suture under such unfavourable circumstances naturally suggests its application more frequently in ordinary cases of cholecystotomy, when the ducts are healthy and are left free of any calculous *débris*, but its obvious risks, and the success of the plan by suture and drainage, have deterred me from risking it in a simple case. It cannot, one would think, be a good thing to have one's gall-bladder fastened to one's peritoneum, and there is the risk of hernia being produced by the drag at the upper angle of the abdominal wound. My next case illustrates this accident, and a case which I have already recorded in the Transactions of the Society shows how adhesion of the fundus of the gall-bladder to neighbouring parts may cause such suffering as to endanger the life of the patient, and closely simulate the most violent attacks of biliary colic. Probably the severe symptoms in this case were due to the adhesions being to tissues free in the abdomen and lower than the natural position of the gall-bladder—it arose from pelvic inflammation spreading upwards—and that as the tissues contracted there was a constant dragging and stretching of the gall-bladder. The absence of unfavourable symptoms when the gall-bladder is sutured into the upper angle of the parietal incision being due to the absence of any drag upon it, its position too tends to keep it empty, while if it is dragged upon by adhesions from below and behind, as in the case referred to, it is elongated and probably always full of fluid. I do not think, however, that in view of the occasional successes of complete suture and dropping, and the occasional hernia following the suture to parietal peritoneum, we can consider the question finally settled as between the two methods.

CASE 3 is that of a lady about 50, who had suffered so much pain for many years from gall-stones that she had become a victim to the morphia habit. She did not suffer from attacks of well marked colic, but had so

much constant and wearing pain in the region of the gall-bladder, that years ago, before these operations were performed, when I was attending her for something else, I told her that I felt sure her gall-bladder must be full of stones and the cause of her pain, and that some day or other she would have to submit to surgery. She was very stout, so that examination of the parts was difficult, and I was never able to convince myself that I could feel either the gall-bladder or anything in it, and I found it at the operation so placed that it was quite impossible ever to have come near it from external palpation. Before submitting to operation she underwent a course of the olive oil treatment, and brought me in triumph a bottle of green gelatinous masses found in the fæces, and said to be gall-stones softened by the oil. I was sceptical, and she became so when it was found that always after the oil about the same quantity of these curious green gems were passed, and in such quantity that a gall-bladder must have been as big as her head to contain them. We then had them carefully examined by two different observers, and they were unhesitatingly pronounced not to be gall-stones at all, but oil acted upon by the biliary acids and contents of the intestines.

I operated on May 11, 1891, in the presence of Dr. Langdon Down and Mr. White, her medical advisers, and found the gall-bladder very deeply placed under the right lobe of a large congested liver, and with the very fat parietes extremely difficult to reach and manipulate. It was distended with fluid, and the cystic duct was blocked with a small angular stone, the fluid contents of the gall-bladder were glairy mucus barely tinged with bile and there were two large roundish stones and several smaller ones in the bladder. I needled up the stone in the duct and then crushed it with finger pressure. This method I now regard as infinitely easier, quicker, safer, and more applicable to all cases than the dilatation of the duct and extraction of the stone, as described in a previous communication to the Society. The liver was so large and congested, and so overlapped the gall-bladder, that it was very difficult to suture the latter into the parietal incision, and I was obliged to use interrupted points. The duct was much thickened and hardened round the stone, and I have no doubt it had been long impacted and was the chief cause of her suffering.

At the end of forty-eight hours I entirely stopped all narcotics, and for some days she was in a very odd mental state, and there was a good deal of depression, but I remained firm in refusing morphia, and during a slow convalescence happily weaned her entirely from this terrible habit, and she is now quite well, but has unfortunately a rupture at the site of fixture of the gall-bladder, which gives so much discomfort that I am going to resect the wound and free the gall-bladder from its attachment to the peritoneum. I have no doubt that the hernia is due to drag upon the peritoneum owing to the peculiarly deep situation of the gall-bladder and the very thick parietes, necessitating an unusually large portion of the upper part of the wound being left to heal by granulation.

CASE 4.—A widow lady, aged 64, had for many years suffered from sharp attacks of biliary colic, and a year before I saw her one of these attacks had been followed by jaundice. From this time the attacks of pain became more frequent, usually about every ten days: there were several hours of severe vomiting and pain, then slight jaundice, followed by a week or so of comparative ease. I found the liver not enlarged, but a large, tender and prominent gall-bladder with considerable hardness and sense of resistance behind and to the left in the situation of the common duct. Her mother had died of some liver trouble at 74, and her only

sister from gall-stones at 38. I operated on June 8, 1891, and removed a large number of variously shaped stones from the gall-bladder, and then found a long irregular stone, not unlike the two top joints of one's little finger, lying in the common duct; it could be slightly moved up and down. I thought it too large to needle and crush successfully, and so I made a longitudinal incision in the duct and extracted it, closing the incision by three fine silk interrupted sutures. I sewed the opening in the gall-bladder into the parietal wound, and at the lower angle of the latter put a glass tube into the peritoneal pouch beside the sutured duct, covering this with the usual rubber sheet and sponge dressing. In twenty-four hours the discharge from this tube was bilious, and there was hardly any from the tube in the gall-bladder, evidently there was leakage from the common duct; finding that this rather increased in a few days, I thought the glass tube most likely pressed against the duct and prevented it from healing, I therefore passed an india-rubber tube through it, and withdrew the glass one over it. After this the bile-stained discharge from the lower tube gradually ceased with increase of discharge from the gall-bladder, and in about ten days I was able to dispense with the lower tube, and she had good bilious actions and the bile began to disappear from the urine; the tube was then withdrawn from the gall-bladder, and the old lady rapidly convalesced, and I have had excellent accounts of her since her return home.

This is another case to add to those already brought before the Society, showing that if asepsis and free drainage are obtained, the passage of bile through the peritoneum is harmless. Though the large stone was soft and friable, I do not think it would have been wise to leave such a long mass of broken-up *débris* in the duct, it would have been very liable to consolidate again and obstruct, and then one would have had a permanent fistula in the gall-bladder. One cannot then lay down any hard-and-fast rule as to when to needle and crush, and when to incise the duct and remove the stone, the operator must exercise his judgment at the time in each case.

CASE 5.—A single lady, just over 40, and apparently in robust health, but suffering from repeated and increasingly frequent attacks of pain in the region of the gall-bladder, was seen in consultation with Dr. Rice Oxley, of Streatham. The diagnosis seemed clear, and the patient was very anxious to have something done, as she had seen her mother die of gall-stone after prolonged and severe suffering. I operated on March 10, of this year, and removed a single barrel-shaped stone without much difficulty from the mouth of the cystic duct. Gall-bladder fixed into parietal wound and drained. There was unusually little discharge in this case from the first and up to the present time she has made a very smooth recovery. The tube was gradually shortened and entirely removed at the end of the week and the wound was healed when I last saw Dr. Oxley, on March 30. The rapid cure in this case shows the advantage of operating before impaction has taken place; the ducts being in a healthy condition, the discharges from the gall-bladder find it more natural to travel down their proper course than up and out through the

tube. The more I see of these cases the more I am convinced that the gall-bladder is not intended to receive bile, but is a secretor of a mucous fluid, and a pump placed on the common duct to flush it out and help to pass on the bile into the intestines.

This completes the list of my gall-stone cases since I last reported to the Society, and happily they have all ended well, as I believe all these cases will when we learn to diagnose them early enough, and operate upon them before serious structural changes have been caused in the bladder and ducts by their presence.

The following case is, I believe, unique, and at one time I hoped might be added to the list of successes in this department of surgery, but its final ending is disappointing and unsatisfactory, in that it leaves us without positive knowledge as to the cause of death.

Early last year Dr. Kraus of Carlsbad wrote that he had recommended a member of our own profession, who had frequently been under his care at Carlsbad for attacks of biliary colic, with only temporary benefit, to place himself under my care for operative treatment. He gave me details of the case, which seemed to point distinctly to its being one of gall-stone colic, and I wrote and advised the patient to come and see me on his return to London. I have unfortunately mislaid my notes of this case, and can only give it from memory, but a personal interview and examination left little doubt on my mind that he was suffering from partially impacted stone. I could not actually detect anything on physical examination, but all his symptoms pointed to attacks of gall-stone colic. I operated on July 30, 1891, and finding nothing in the gall-bladder, passed my finger down the outside of the cystic duct, and near its entrance into the common duct, found a hard swelling of somewhat angular form, which felt exactly like an impacted stone; failing to move it either up or down, I passed a needle into it through the duct wall, and then at once felt that I had not to do with a stone, but with a solid growth. I divided the wall of the duct longitudinally, freely enough to get my finger round the growth, and found that it projected into the duct, and had a somewhat broad base; with my nail I soon enucleated this base, and it shelled out in such a manner as to give the impression that it was more or less encapsuled; there was no serious hæmorrhage, and I closed the opening in the duct with a few points of interrupted suture. I then found immediately behind the duct an enlarged gland, which I also removed. I could find nothing else ab-

normal, though I carefully palpated all the parts. On section the growth appeared to me to be sarcomatous, and I decided to remove the gall-bladder, and tie the duct just above my wound in it. I was induced to remove the gall-bladder by the consideration that there would be less risk of recurrence if the function of the part was abrogated, and the duct allowed to shrivel up. He made an ordinary recovery, except that he appeared to suffer a great deal more than the usual amount of pain; but knowing that he had contracted the morphia habit strongly, I did not attach much importance to his complaints, when he had been so recently largely deprived of this drug. He was also very difficult to feed, and suffered much from nausea and sickness, which I also attributed chiefly to his morphia habit. The glass tube was removed from the peritoneum within a few days after the operation, and the rubber tube which replaced it was in for about a month; but Mr. Malcolm, who kindly took charge of him for me after the operation, thought it might have been removed before it was, as the wound healed at once without further discharge. As in some of the other cases in which I have incised the ducts, the union was not immediate, as shown by the escape of bile-stained fluid from the peritoneum for some time after the operation. There was enough discharge for the first two weeks to make it necessary for Mr. Malcolm to dress him night and morning, after that a daily dressing sufficed till the rubber tube was finally removed, when the wound at once healed entirely. He left London six weeks after the operation. Though he thus made a satisfactory surgical recovery, there were all through differences in his progress, and that of a well-doing gall-stone case. I do not think he was ever entirely free from pain, and his appetite was poor and capricious, and on two or three occasions he coughed up a little blood; there was a strong suspicion, however, that this was from the throat only, and caused by his persistent retching and coughing. The great difficulty was to differentiate between actual pain and discomfort, and the irritability, moral and physical, of the morphia habit; so far as pulse and temperature and condition of intestinal and urinary secretions could guide, he made a very good recovery. He went to Dover on leaving town, and then to Canterbury, where he was under the care of Dr. Gogarty, who had known him and attended him occasionally for some years. Dr. Gogarty wrote on October 24th, "The pain is as bad as ever; the attacks are similar to those he

had before the operation; he has lost flesh considerably, and the superficial abdominal veins are enlarged, but no tumour can be felt; the pain is not localised, but is referred generally to upper part of the abdomen and left side of chest, where there is dulness, not due, I think, to fluid, though there may be some. He has been using morphia hypodermically rather too freely in my opinion, and it has set up irritability of the stomach and distaste for food. The pain, nausea, and inability to take food are rapidly wearing him out. It is undoubtedly an obscure case, but I am inclined to think it is an aneurism." Two days later Dr. Gogarty wrote announcing his death, and that he could not obtain a *post-mortem*. The growth was a small round cell sarcoma, and the gland removed at the same time was quite healthy. My own impression is that there was some rapid recurrence, as evidenced by the pain, rapid emaciation, and fulness of the superficial abdominal veins. I have not been able to find any record of a similar case, and I shall be greatly obliged to any Fellow of the Society who can direct my attention to any recorded case, if such an one exists. I cannot see any good ground for thinking that he suffered from aneurism, and I feel sure that I should have detected it if it was abdominal, or, as Dr. Gogarty thought, at the entrance of the aorta into the abdomen, for I made a most careful examination of all the parts at the time of the operation.

If such another case should ever come under my care, I should dissect out the whole of the duct at the seat of disease. The connective tissue, muscular tissue, and mucous membrane round the growth were quite healthy. It occurred to me that a small angular stone might have been impacted there, set up new growth, and itself become dislodged and disappeared. I on one occasion found a small angular stone completely embedded in the wall of the gall-bladder.

Two other cases to which I will briefly refer complete my operative work in this department of surgery since my last communication to the Society. In January of last year I saw, with Mr. Stormont Murray, a young married lady who was suffering from frequent attacks of what appeared to be biliary colic; she had had jaundice from cold at 13, complete amenorrhœa from chill for six months at 15, and the attacks for which I was consulted began at 16, the pain would come on suddenly, night or day, in the epigastrium, last a variable time, and pass off gradually round right

side, and across the back; an attack very often preceded the menstrual flow. I did not think the attacks were due to gall-stones, but thought there might be some contraction of the ducts from the catarrhal attack in early life. No improvement took place, and the attacks being very frequent and troublesome, it was decided two months later that I should explore. I made a small incision over the gall-bladder on February 26th, found nothing palpably wrong with either the gall-bladder, its ducts, or surroundings, and closed the incision. She made a perfectly smooth recovery, and if no better was certainly no worse for the operation.

About eighteen months back I saw, with Mr. Venning, a gentleman of about 30 years of age, who was suffering from attacks of pain in the epigastrium with a distinct tender swelling in the region of the gall-bladder. I examined him carefully; could not satisfy myself as to the nature of the swelling, or that the attacks of pain were gall-stone colic, and did not feel justified in recommending any surgical interference. His attacks of pain continued; riding, running, or any jolting movement making them worse, so that he found himself unfit for his duties as an officer in the army, and he consulted Dr. Broadbent, who thought there was enough suspicion of the case being one of gall-stones to make an exploration advisable. I was accordingly again consulted, and finding a very definite swelling, which I thought might possibly be hydatid, I consented to operate. When he was well under the influence of chloroform, the very definite rounded swelling over the site of the gall-bladder entirely disappeared, so that it was a muscular phantom raised at the will of the patient or involuntarily to protect the painful part. This is the only instance of this kind of phantom tumour that I have seen in the male. The exploration showed that the gall-bladder and ducts were healthy, but that there was a considerable and somewhat nodular enlargement of the left lobe of the liver. The patient had at one time been a heavy drinker, and had also had syphilis, so that it remained uncertain to which of these evils the disease was due. He made a rapid and smooth recovery from the exploration, but was distinctly jaundiced for a few days. We decided to put him on a full course of iodide of potassium, in the hope that a like satisfactory result to that related in my previous communication in a similar case might follow. The other patient is quite well, and I hope that I may be able to report favourably on the present case at some future time.

I will now briefly refer to a few points which are, I think, clearly demonstrated in the series of cases I have from time to time brought before the Society.

1. A properly-conducted exploratory incision is free from risk, and may give valuable information leading to a scientific and successful after-treatment, even in cases in which no stone is found. Such exploration is, however, only justifiable after the most thorough trial of dietetic and medicinal treatment.

2. The ducts are completely within the sphere of successful manipulative and operative measures, when such are needful, as is the gall-bladder itself.

3. Stones of moderate size impacted in the ducts should be treated by needling and crushing, either by finger and thumb or by forceps-pressure. Larger stones by incision, complete removal, and after-suture of the duct wall.

4. In cases in which *débris* is left in the ducts, the gall-bladder should be opened, sutured into the abdominal incision, and drained.

5. In cases in which the ducts are incised and sutured, a drainage tube should be passed into the peritoneum beside the sutured duct, and the gall-bladder also drained externally.

6. In a well-marked case of repeated attacks of gall-stone colic with recurring distension of the gall-bladder, it is better to operate early, and before there is a chance of injury to the duct wall from impaction, and before the stone has reached the common duct. Such cases recover rapidly, and there is every prospect that experience will show that complete intraperitoneal suture will be safe and the rule of the future when operation precedes damage to the ducts from impaction.

In conclusion, I have only to repeat what I have said before, that in this as in other branches of abdominal surgery, what we want is more perfect diagnosis. Let me then urge both the physician and the surgeon to note most carefully for themselves the actual symptoms, not trusting much to the statements made by the patients, who are generally in too much suffering to be very accurate in their recollections. I have no faith in either olive oil or yellow soap, and not much in courses at Carlsbad and similar places, but I am sure an immense deal of acute suffering will be avoided, and many valuable lives will be saved by a prompt and skilful use of the knife for the removal of gall-stones.

Practice shows that they can be removed with safety and without suffering, why then should we hesitate, when at the best they can only be got rid of through the ducts with much and prolonged suffering and serious risk. We should, without hesitation, remove irritating foreign bodies from other tissues and situations, why not gall-stones ?

Dr. DOUGLAS POWELL said that the needling and manipulation of a stone in the duct were to him a novel procedure. Mr. Thornton seemed to regard the gall-bladder not as a reservoir for bile, but as a receptacle of fluid with which to wash out the bile-duct. He himself would like further evidence in support of this before accepting it, for it certainly impaired the value of some experiments which had been made as to the secreting function of the liver, seeing that the material used had been derived from the gall-bladder.

Dr. RALFE, though he admitted that surgeons had relieved physicians of many of these troublesome cases, yet held that there were instances in which it would be better if they waited longer before operating. In one case in which cholecystotomy was done, the gall-bladder was found empty and cancer discovered to be present. In another case, in which operation was urgently recommended, the patient got well with rest and the use of mineral waters. Many attacks of so-called gall-stone colic were spurious and due to other causes, such as a collection of scybala at the hepatic flexure of the colon ; or, as in one instance, the presence of a large round worm. Medical treatment might certainly be carefully tried before operative interference was thought of.

Mr. KEETLEY had operated on a middle-aged man who, for six months previously, had suffered from a frequent succession of attacks of biliary colic, and had become greatly wasted. The gall-bladder was found small, thick-walled, and apparently empty, and as no gall-stones could be felt either in the bladder or the ducts, and as it would have been difficult in this case to bring the gall-bladder into contact with the parietal perinæum, nothing further was done, and the wound closed. But from that time the patient rapidly improved, had no more attack of colic, and in a few months was in robust health, in which he remains. Whatever other causes might lead to hernia after an abdominal section, one essential factor was imperfect union or non-union of the different aponeurotic layers and strong sheets of fascia each to each.

Dr. BRISTOWE regretted that he had only arrived in time to hear the latter part of Mr. Thornton's paper, for the subject-matter of it was one that interested him very greatly. He had listened with pleasure, however, to the latter portion of it, and agreed substantially with the conclusions which Mr. Thornton had formulated. He did not himself believe that medicines had any influence over gall-stones, excepting in so far as they relieved pain and other sufferings induced by these bodies and relaxed spasm. And he had no doubt of the great benefit to be derived from surgical treatment judiciously applied. He did not look on exploratory operations as wholly free from danger, or as justifiable as a routine treatment. For he regarded the passage of gall-stones as being an extremely common incident, far more common indeed than most persons supposed, and as usually unattended with any serious risk ; and his own experience had shown him, that operations were occasionally performed (no doubt on sufficient clinical grounds) without anything being discovered to

justify them, and that occasionally they led to fatal results. With respect to the frequent occurrence of biliary colic, he might mention that some fifteen or twenty years ago he had within a short time of one another three patients, varying in ages between 18 and 21, who were admitted into St. Thomas's Hospital suffering from attacks of some "gastrodynia," coming on suddenly, attended with vomiting, and subsiding after a few hours or a day or two. The patients had no jaundice, and in the intervals appeared to be well. He observed at the time that the symptoms were like those due to the passage of gall-stones, but that the ages of the patients were adverse to that view. The sister of the ward, who was a very intelligent woman, acted on the hint given her, and in each case succeeded in discovering a gall-stone, which she handed to Dr. Bristowe at subsequent visits. One of the patients was afterwards admitted with jaundice, following one of his attacks. Since then Dr. Bristowe had seen many cases even in young persons, in which he had been able to refer to gall-stones, attacks of "gastrodynia," "gastralgia," "spasms," or "colic," which had been supposed to be due to "indigestion," to "error of diet," to "gout in the stomach," or other imaginary disorders. It is obvious that in such cases as these no operation is called for or would be justifiable. It is interesting, indeed, to know that gall-stones occasionally obstruct the common duct for long periods without causing serious organic disturbances. Some years ago Dr. Bristowe saw an elderly gentleman who, after an attack of biliary colic, had remained jaundiced and without any passage of bile into his bowels for some two or three months. He subsequently learnt from the patient's son, who was a young physician, that he had continued in this condition for twelve months, that then he had a recurrence of colic, followed by the passage of a gall-stone (which he secured), subsidence of jaundice, and restoration to health. He did not quote the case as an argument against the resort to surgical treatment in such cases. It occurred before hepatic surgery had come into use. If such a case came under his care now he should urge an operation. That operations may be performed to cure what does not exist is a contingency for which one must be prepared, and is a reason for caution. Some two or three years ago Dr. Bristowe was consulted in the case of a lady who had apparently had repeated attacks of gall-stones, and when life had been rendered thereby a burden to her. When he saw her she had recently recovered from a severe attack and seemed well. But it was thought desirable, looking to her past history, and to prevent recurrence, that an exploratory operation should be performed, and in this view he concurred. The operation was performed, but there were no gall-stones found or other evidence of disease. He knew nothing of her later history, and was not prepared to admit that the operation was unjustifiable. That such operations may fail of their object, and are attended with risk to life, is shown by a case that was under his care a short time ago, and was published by his friend and pupil, Dr. Copeman. A young woman came under his care suffering for complete obstruction of the common duct of some two or three months' duration. The case was rather obscure; but he leant to the diagnosis of obstruction of the duct by a gall-stone; and as the case presented very grave symptoms, which showed no signs of amendment, it was decided, after consultation with one of his surgical colleagues, that an operation should be performed. This was done; the gall-bladder was laid open and discharged a quantity of colourless glairy fluid, but no gall-stones were detected either in it or in the duct. In the course of a few days bile began to flow from the gall-

bladder, and before long the escape was free. The patient's jaundice disappeared wholly, and her health improved marvellously, but still no bile found its way into the bowels. On one or two occasions an attempt was made to pass a probe from the gall-bladder to the bowel, but without success. Subsequently profuse hæmorrhage took place from the bladder, and this proved fatal. At the *post-mortem* examination a small calculus was found in the common duct just below the orifice of the cystic duct. It may be added that before the parts were dissected out they were carefully examined with the object of ascertaining whether any cause of obstruction could be felt; no stone was felt. In conclusion, Dr. Bristowe adverted to one of Mr. Thornton's cases, of which he happened to know something. It was that of a young medical man, who had formerly been one of his pupils. He had consulted Dr. Bristowe before submitting to abdominal section, mainly with the object of knowing whether Dr. Bristowe considered the condition of the heart such as to allow of the safe use of anæsthetics, for he had formerly had rheumatism, and there were evidences of slight valvular defect. Dr. Bristowe, on that occasion, knowing what was contemplated, took the opportunity of examining him very carefully, and he does not hesitate to say that there was no suspicion at that time of abdominal aneurysm or of any other abdominal tumour; and that, with Mr. Thornton, he entirely disbelieved that he had any aneurysm.

Mr. MARMADUKE SHEILD said that in the diagnosis of smaller abdominal tumours, such as gall-stone or movable kidney, it was necessary to submit the patients to a preliminary and thorough course of purgation. Gall-stones often existed without causing any symptom, and were very frequently found *post mortem* where they were by no means suspected. Abscesses of obscure formation, pointing in the right iliac fossa, at the umbilicus, or through the abdominal wall, often when they burst discharged a quantity of gall-stones. The case related, in which healing took place in spite of the fact that malignant disease of the gall-bladder was present, was interesting; but bones which fractured as the result of malignant disease would firmly unite, and so would the cut surfaces of a peritoneum which had undergone malignant infiltration. He asked what was the value of massage in cases of impacted gall-stone.

The PRESIDENT said that he could not accept the theory which had been advanced as to the function of the gall-bladder, for that structure in the foetus was proportionally so very large. Although the Society highly appreciated the successes that had been recorded, yet he felt bound to enter a caution against any attempt at imitating these procedures among the profession generally. The public ought not to be taught to look upon gall-stones as substances which, like urinary calculi, when found ought to be removed. They were constantly found, for instance, in lunatics, especially melancholiacs, with no symptoms whatever. It was not the mere recognition of stones in the gall-bladder, but the sufferings which patients endured from them, that should be the measure of the necessity for operation, and in this respect there was an essential difference between gall and urinary calculi; besides, they were all aware of cases which after operation had not proceeded to so happy a termination as those of the author seemed to have done. Perhaps a little later he would supplement the procedure of crushing the stone by flushing and washing out the fragments. In conclusion, he narrated the history of two females who had had gall-stones for as long as twenty years, and though they had suffered much from them at first, yet at present the symptoms had entirely subsided, although the calculi were still to be felt.

MR. KNOWSLEY THORNTON, in reply, said that in his paper he had carefully defended himself from the assertion that he desired to operate on all biliary calculi. Though he agreed that in many cases they did no harm, yet against such cases were to be put others in which perforation and gangrene of the duct occurred and the patient died in a few hours. It would be difficult to evacuate the crushed calculi, because one was dealing with a closed duct through which nothing could be washed, and which was therefore quite unlike a pervious urethra. He agreed that a thorough clearance of the intestines was necessary before arriving at a diagnosis. There was often no jaundice and no vomiting until the stone arrived in the common duct ; but the case might prove fatal before this happened. He could not recommend massage as a method of treatment in these cases, for in two in which it was resorted to, he found on operation that the stone had grown into the duct wall, and manipulation from without could only have ruptured the duct. Though occasionally cases could be met with which, after twelve months, had come to such a happy termination as that related by Dr. Bristowe, yet he doubted whether it would be right for a surgeon to wait beside a jaundiced patient for twelve months for the possibility of the stone moving. He related an instance in which he was unable to feel the presence of a stone which he afterwards found on opening the gall-bladder.

April 25th, 1892.

ON SOME OF THE RARER COMPLICATIONS OF RHEUMATOID ARTHRITIS.

By JOHN KENT SPENDER, M.D. Lond.

THE arthritic phenomena of the disease officially known as osteo-arthritis, but more commonly as rheumatoid arthritis, have received so much attention that we are apt to forget the kindred symptoms which are the frequent accompaniments of the disease. It sounds bold to speak of rheumatoid arthritis as simply a grand neurosis, with arthritis as a collateral fact, and sometimes not at all the most important. But the careful study of rheumatoid arthritis on a large scale, and a comparison of the different clinical types on which it is evolved, will display a variety little suspected by those who see little of it.

A primary axiom of my subject, then, is that the earliest symptoms of rheumatoid arthritis are not necessarily arthritic at all. We see sometimes a complex state in which the neural and trophic phenomena of the body are strangely upset and confused. What

can it be? What does it all mean? The heart beats with unusual quickness and force; the skin is becoming darker here and there; complaint is made of paroxysmal sweating and pain; scarcely anything, perhaps, is said about the joints. We are haunted by visions of central and peripheral disturbance. We talk vaguely, it may be, of suppressed gout—that refuge of “feeble diagnostic power”—and in a puzzled way we pass in review all sorts of conditions, diathetic and other. At length we find a clue, which in the old days would have been “rheumatic gout;” in chronological order it became “rheumatoid arthritis,” and then it enjoyed the official style of “osteo-arthritis.” Now we are waiting for a title which, while strictly connoting the arthritic part of the trouble, shall at least hint at the grave dynamic commotion which often accompanies it, and helps to give it a characteristic outward expression.

At the outset of our inquiry we will eliminate altogether that special arthritis which is a pure senile degeneration—the hip-ache and disability of elderly people. In the esoteric terminology of our Mineral Water Hospital we exclude such cases from the group of true rheumatoidal lesions, and look at them just as we look at cataract or white hair. Dr. Archibald Garrod calls this arthritis a “localised variety” of rheumatoid arthritis; but the facts that it is often non-symmetrical, and can be generally traced to a local injury, seem to justify a separate classification entirely. And for other and sufficient reasons we put aside all the forms of septic arthritis to which attention has been called by Dr. Edward Blake.

It is unspeakably dreary to intrude into a short paper the narrative of a long and complex case. Suffer me, however, without transgressing my own canon, to begin my subject by relating in the most compendious way the bare outlines of a case quite extraordinary in its power of lightening up some dark corners in the neurology of rheumatoid arthritis.

Early in June, 1891, a lady, aged 43, was entrusted to my care by Mrs. Louisa Atkins, M.D., after a consultation with Sir Andrew Clark, at which a diagnosis was made of rheumatoid arthritis in its early stage. It was determined to send her to Bath; and, when I saw her, there could be no doubt of the anatomical and clinical facts. But soon after her arrival in Bath and a short thermal treatment, the rheumatoidal part of her malady seemed to wane. Other and more serious symptoms came forward—“bulbar warnings,” as I called them in a monograph three years ago. These warnings included a feeble action of the masseter and

pterygoid muscles, so that mastication and swallowing were imperfectly performed ; a difficulty in protruding the tongue ; an altered and sluggish expression of the face ; and a progressive muscular atrophy of the arms, shoulders, neck, chest, and intercostals. An appointment to see Dr. Buzzard was made for July 15th ; and in the presence of Mrs. Atkins he examined our patient in the most thorough manner. The resources of electro-diagnosis were employed ; and he confirmed our worst fears by a declaration of polio-myelitis of the cervico-dorsal region of the spinal cord, extending to the motor nuclei in the medulla oblongata of the ninth nerve, of the portio dura of the seventh nerve, and the root of the spinal accessory. The prognosis was very grave. Still, we did not surrender the hope that these bulbar complications might pass away, and that obstinate functional inco-ordination had not yet ripened into incurable disease. As a matter of fact, the power of swallowing did return to a certain extent. But further degenerative changes soon occurred. Visceral lesions threatened life, and our patient's local medical adviser desired to have the judgment of Dr. Suckling, of Birmingham. After a careful investigation, Dr. Suckling gave the opinion that her symptoms proceeded mainly from sclerodermia. Death ended much suffering in the third week of January, 1892.

This patient was, you see, studied by a number of competent observers. Their views of her pathology were different, but not necessarily opposing ; and the opinions formed depended upon the stage at which the case was seen. It was like a long procession, observed by one man at one point and by another man at another. Each reports what has passed under his own eye. If no observer was entirely right, certainly none was in the least wrong. The whole history was homogeneous and consistent. The morbid anatomy of joints was the initial sign of a profound nerve disturbance, recognised by Sir Andrew Clark and Mrs. Atkins in such a manner that the lady was sent to me as an example of pure rheumatoid arthritis. But to Dr. Buzzard the arthritis had so receded into the background that, to quote from his letter to me, "I have to depend upon your description of the articular symptoms, for the muscular symptoms quite overshadow them. I find neither swelling, nor pain, nor crackling." And finally, Dr. Suckling discovered only atrophy of skin and connective tissue, which in its turn had overshadowed arthritis and muscular wasting. Does not this sequence of events justify the assumption which I ventured to make at the beginning of my paper, that what we call rheumatoid arthritis is a far-reaching neurosis, having a variable etiology, and co-extensive with the forms and functions of many organs ?

I now propose to speak in an orderly way of some extreme types of disorder which may be associated with rheumatoid arth-

ritis. I lay emphasis on the words *extreme* and *unusual*, with the object of narrowing and making more vivid the scope of my paper.

1. The circulation is disturbed in such a way as to suggest that the excitability of the heart is no longer controlled by vagus action, to express ourselves in the terms of Professor Roy and Dr. Adami in their recent contribution to the Royal Society.* Or the fact might be expressed in the old manner, that the inhibitive or restraining force of the vagus nerve was not exercised in the usual and physiological way. Putting aside all moderate and comparatively common cases, I refer now to those rare instances in which the pulse rate is always above 120. In 1887 a young lady (living at Wincanton) was under my care for some weeks in Bath; she had a permanent pulse of 140 in the minute, and its tension was so high as to shake her whole body. A man lately in our Mineral Water Hospital had a steady hard pulse of 132. In neither of these patients was the rheumatoidal lesion obtrusive or severe. They had no physical signs of dilatation of the heart or of damage in any of its valves. There was no palpitation in the ordinary sense of the word, and no interruption in the regular march of beat upon beat. When the cardiac action reaches this point of sustained hurry, I have found no remedy of any material use; but in the minor forms (if I may so style them) of rapid pulse there is often a marked decrease of rate and tension as the health improves.†

2. There are two extreme forms of pigmentation. (a) The melasma is so like suprarenal discolourment that the first glance of a patient so disfigured reminds one of Addison's disease. Wrinkles on the forehead are white furrows parallel with so many dark ridges; and the skin of the neck looks as if soaked through with a walnut dye. It is analogous to the thyroïdal bronzing described by Dr. Drummond. (b) The multiple xanthoma of far advanced rheumatoid arthritis is decidedly rare. I have already published some account of a lady of middle age (under my care from 1884 to 1886) who had yellow patches and rashes on a large part of her body. The exceptional point in her case was the

* Reprinted in the 'British Medical Journal,' February 27th, 1892.

† My original observations on the special quick pulse of rheumatoid arthritis have been confirmed by several physicians, among whom are Sir Dyce Duckworth, Dr. Sansom, and Dr. Archibald Garrod.

separation and twisting of most of the nails of fingers and toes from the matrix by an accumulation of dry, chalky material. But circlets of a bright yellow tinge are not uncommon around the finger joints on their dorsal surface. (c) A curious complication is the coming and going of small areas like bruises on a rheumatoidal limb. They pass through the same sequence of colours as a bruise. So exactly is the "bruised" area like the result of an ordinary contusion, that no other comparison would suggest itself to anyone who saw it for the first time. In an elderly lady sent to me by Dr. Lewis, of Folkstone, and in a lady of middle age, sent by Dr. Gray, of Oxford, the rather sudden development of these chromatic patches determined the nature of each case beyond dispute. They come without warning, and even without the patient's knowledge. They are mostly of about the diameter of a florin, not raised, and they can be pressed without eliciting any sensation like pain. The tracks of nerves or of blood vessels cannot be associated with them. In my last example of this strange phenomenon the discoloration was purple-black at its height, and then gradually subsided through a procession of hues to a light dingy brown.

3. The common atrophy of muscle and skin which goes along with rheumatoid arthritis has been described by many writers, notably by Dr. Ord and Dr. Archibald Garrod; and it is my business here to describe only those errant phases of motor function which are eccentric and rare. A "to-and-fro spasm" of one arm, of the strict shaking palsy kind, has been observed by me in two cases in which the rheumatoidal lesion was confined very much (though not entirely) to the shaking limb. An extremely cold, damp hand, purple-blue in tint, and with the distinctive feature of glossy skin, may display a curious mimicry of athetotic movement so far that the fingers do not bend or extend in a harmonious and coherent way. And I have seen the fingers engaged in slow, involuntary movements when the attention has been directed elsewhere.

4. The neuralgia which is such a frequent companion of rheumatoid arthritis deserves special note, because it is so commonly misunderstood. As an early symptom its value is scarcely recognised yet. Take an actual instance: A lady in middle life has for a period of six weeks an acute pain in the region of the right shoulder. It is always there—often it is worse at night; but for

awhile there is no paresis of muscle or impediment to free motion of the shoulder-joint. The cause of the pain is obscure; no medical advice is sought, and nothing definite is done. Gradually the arm cannot be lifted; even passive movement cannot raise it beyond a certain angle. Before skilled care is bestowed upon the case, the first stage of rheumatoidal change has come and gone; alteration in the synovial secretion and some adhesion between the joint ends of the bones are accomplished facts; and there is distinct atrophy of muscle around shoulder and upper arm. A mere surface study of this brief history might suggest that "rheumatism" of the shoulder was the sole cause of the pain; that it was, in short, a rheumatic pain pure and simple. I believe that the sequence of events was precisely the other way, and this is my reading of them: Something wrong began in the lateral sensory column of the spinal cord; a dynamic irritation was set up in the cervical plexus, as expressed by the pain; and this perversion of molecular energy resulted, after a time, in actual morbid change. Why the stress of that change should fall upon the first joint which the nerves reach is beyond our present knowledge; but so it is.

The symptom of pain is often (but by no means always) in inverse proportion to the weakness. This is my strong impression; but it is not easy to state it as a formula because, although we can measure weakness by specific tests, we have to depend upon the oral statement of a patient as to the degree of pain. It may be so severe or persistent as to be *the* thing complained of. Thus, a sciatica is talked more about than a rheumatoidal hip or knee; but the neuralgia and the lame joints are the common and contemporaneous effects of a dynamic irritation of the lumbar and sacral plexus of nerves. When neuralgia haunts a limb, and no likely cause (such as malign disease) can be detected, we may suspect rheumatoidal degeneration in the proximal and larger joints.

This phenomenon of pain marks an intimate kinship between rheumatoid arthritis and locomotor ataxy. I have mislaid the reference to a case published in a medical journal within the last few years, the record of which stated that an injury to the nerves of the upper arm was followed by an arthritis of the elbow which closely mimicked a rheumatoidal lesion.

Am I straining analogies and alliances too far in thinking that

there may be a subtle link between rheumatoid arthritis and osteitis deformans? A rheumatoidal lady now under my care has her right collar bone big and misshapen. The external or flat segment is as much distinguishable from the internal or curved segment as if there had been a fracture, and the separated fragments of bone had united in an irregular and clumsy way.

5. Vasomotor derangements of an extreme type are exhibited now and then in the hands and feet. Dampness and wetness are common enough; but it is not very infrequent for the hands to stream with perspiration as if just immersed in water. The long trough in the situation of the vertebræ may be a canal of running moisture. No symptom is more unerringly diagnostic than this. A fresh patient is admitted into our Mineral Water Hospital, and we are for a moment in doubt about the nature of the arthritis. If the hands do not tell the tale plainly, we turn up the bedclothes and see the dew on the feet, and the problem is solved beyond debate; and a purple blue hand, frigid on even a hot summer's day, may show a mottled finger so dark in tint as to remind one of the vagaries of Raynaud's disease.

My paper may fitly close with a reference to one or two subordinate points.

The researches of Westphal on the neuritis which occasionally succeeds influenza must not be passed over. He speaks of coldness and numbness in fingers and toes, weakness and wasting of muscles in both upper and lower limbs, paresis of groups of muscles, and pain on pressure over nerve trunks and muscles.* These are the very symptoms which, with an undoubted arthritis, I have seen again and again as the immediate and remote sequel of influenza, the influenza being in several instances mentioned by patients themselves as a probable cause of their rheumatoidal lesions.

I plead that the simple and useful instrument, the dynamometer, is not applied nearly so much as it ought to be. Imagine if you will that a rheumatic or gouty pyrexia and weakness have passed away, and that the hands have recovered their pristine power. Soon afterwards, and quite unexpectedly, a new weakness and a new pain are complained of. What does this signify? The first thought may be—it is only the dregs of the old illness, the paresis which comes from waste and inaction. How is this to be

* Quoted in the 'Lancet,' January 10th, 1891.

determined? By the dynamometer and that alone. Put it into the grip of the damp, flabby hand; coax the hand how you will, and the failure of force is often not merely relative but absolute. We are almost shocked by finding that the index only quivers on the dial. Rheumatoidal atrophy has already begun. Perhaps the hand looks big and almost clumsy, quasi-potential in substance and form; but the deception comes from the enlarged joint-ends of the metacarpal bones, the spindle-shaped interdigital joints, and the thick carpal region of the wrist. The minatory clauses will soon be there—the withered strands, the shrunken cushions of muscle, the hollow interosseous spaces.

I am compelled by sheer lack of time to pass over the visceral complications of rheumatoid arthritis. Probably it will be demonstrated soon that the sympathetic system of nerves shares largely in the neural shock of the disease. I have referred elsewhere to the “gastric crises” which mark an affinity between rheumatoid arthritis and locomotor ataxy; and Mr. Maude has shown that this is an occasional symptom of thyroïdal enlargement.* The subject needs a most careful investigation, and the help of many observers.

I conclude with certain propositions which may be at least working hypotheses towards that fuller knowledge which we all desire. There is a mine of as yet almost unexplored treasure.

(a.) In its history and symptoms rheumatoid arthritis has distinctive notes of being under the dominion of the nervous system.

(b.) It may receive an initial impulse from rheumatism or gout, and possibly from other diathetic conditions. All causes of nerve depression and blood deterioration favour the rheumatoidal state.

(c.) When fully developed it is itself the cause of an “arthritic cachexia,”—to borrow an expressive phrase from Sir Dyce Duckworth.

(d.) This cachexia may be associated with profound lesions of nutrition, with pain, with atrophy, with errors of inhibitive control, and with even visceral disease.

(e.) Amelioration, and perhaps even cure, are possible in the earliest stages; but beyond a certain point, rheumatoid arthritis is as unmanageable as if it belonged to a malignant nosology.

* ‘Practitioner,’ September, 1891.

The PRESIDENT questioned the advisability of using the term "rheumatoid arthritis," and he thought that some of the cases quoted as examples of complications were really instances in which other maladies joined with the arthritis in producing a multiple disorder. The vasomotor system took a share in the production of almost all diseases, but it was excessively difficult to say whether it exerted the primal influence in departure from health. He believed that the old term "rheumatic gout" was still the best for this group of cases; on the rheumatic side it proceeded from a nervous, and on the gouty side from a blood origin.

Dr. ORD thanked the author for bringing forward his interesting considerations regarding osteo-arthritis, which, however, he preferred to regard not as an objective disease, but as a symptom belonging to a number of conditions; whether we were studying or treating the malady, the latter was the most philosophical standpoint. This characteristic condition of osteo-arthritis, whether overgrowth or atrophy, was met with in varying conditions; in gout, after acute rheumatism, in affections of the nervous system, either as a direct sequence of lesion in the anterior horns of the cord, in pachymeningitis, peripheral neuritis, or lead poisoning, or in association with internal mischiefs not originally of nervous origin, such as congestive uterine affections, &c., in lesions of the prostate and adjacent urethra. It might be associated with injury of a joint, either sudden, as a blow, or more gradual, as in chronic strain, or with injuries of bones near joints, the latter not being involved in the injury. He was not prepared to say how far sepsis should be included as a cause. The first thing was to recognise that the joint affection might occur in a large number of conditions, and then so far as possible to recognise the conditions which surrounded each. In the case quoted by the author at length there was the osteo-arthritis followed by the spinal trouble and the so-called scleroderma, but this sequence had often been noticed in cases of chronic anterior poliomyelitis and of chronic spinal pachymeningitis, and in peripheral neuritis likewise. In conclusion, he thanked the author for the interesting account he had given of such correlative symptoms as abnormal pigmentation and local perspirations.

Sir DYCE DUCKWORTH remarked that the whole subject under discussion was so large that he would confine what he had to state to a confirmation of the arguments which appeared to justify the view that the nervous system was largely involved in the origin and evolution of chronic rheumatic arthritis. He agreed entirely with the views expressed by Dr. Spender, and also accepted those of Dr. Ord, which enlarged one's conception of this disease, and which clinical study tended to confirm. He believed that on no other hypothesis could many of the leading symptoms of the disease be explained. When we considered that the peculiar pains, the interference with cardiac rhythm, the sweatings, the pigmentary changes were definite and specific, though not all of them constant features, we were compelled to recognise the predominant nervous element in the disease. The trophic changes were not merely senile, but indicative of central nervous degenerative lesions which might, in time, come to be ranged with a large class of allied tropho-neuroses, expressed variously perhaps, as in the case of Charcot's disease of joints, but still significant of central degenerative changes. He had hoped to hear more in Dr. Spender's paper respecting the therapeutic measure found useful in his experience at a station like Bath, and urged that in our minute studies of pathology we could not, as physicians, forget that our business was primarily, if possible, to heal the sick.

Dr. EDWARD BLAKE was of opinion that little would be done for rheumatic gout, until much more is known about its causation. As a contribution to its etiology, Dr. Hughlings Jackson had recently made a most valuable suggestion. Dr. H. Jackson had hinted at the possibility of the products of katabolism being the normal stimuli of the circulation. Of course that is when they are present in limited quantities. If this be the case, we get a new reason why exercise stimulates the heart. Uric acid is supposed to possess this property. Alcohol and the soluble salts of lead are said to share this power. Various septic products are well known to be able to accelerate the heart. But all these agents are credited with the ability to set up arthropathies. So we may venture to add to the sage suggestion of Dr. Hughlings Jackson, the proposition that the agents which accelerate the heart may induce chondro-synovitis or else myalgia. But they do not cause gout or rheumatism in every instance. The exceptions may be explained in the following way. I have observed that, during active sexual life, these agencies, including traumatism, shock, innutrition, hot weather, arsenic, the toxins of miasm, and of influenza, are prone to produce a neurosis or a psychosis instead of a joint or muscle lesion. This appears to hold good of women more than of men. About two years ago, Dr. Sansom made a most interesting contribution to the proceedings of this Society, on Tachycardia or heart-hurry. It is suggestive that his concomitants of persistent palpitation constituted a fairly complete group of passive septic invasions. The practical outcome of this is the golden rule: when embarrassed by an obstinate arthropathy or by an intractable neuro-psychosis, we should hunt for latent pus, arsenic, lead, or other distinct toxic agencies.

Dr. SANSOM, referring to the Presidential remarks, said that undoubtedly the term "rheumatic gout" was good in a certain class of cases in which the symptoms of both were commingled, but those were not under discussion in the paper. The author was referring to such cases as the following:—A middle-aged woman would present vague symptoms, palpitations, gastric crises, and anomalous kinds of pain here and there; then changes in the joints might be found, such as thickenings of the knuckles in which there was no pain. One then went more deeply into the case, and found that the heart was unusually quick—not paroxysmally but constantly, the condition persisting even for years; in other cases it was markedly irregular; in all, arthritic phenomena would be found unaccompanied by uratic deposits. Such cases were a strong argument in favour of a change in the central nervous system. It was unlikely that the cardiac condition was due to an irritation of the accelerator mechanism, but it probably followed an alteration of the relation between the vagus and the sympathetic, and the cardiac action thus resembled the exaggerated reflexes seen when certain parts of the nervous system were cut off from the control of the higher centres. The term "osteo-arthritis" might not be perfectly well chosen, but it served to indicate the joint lesions which followed changes in the central nervous system.

Dr. ARCHIBALD GARROD agreed that the ulceration of cartilage, &c., should not be looked upon as a disease, but as a symptom; and the various conditions which the author had enumerated as complications should rather be looked upon as morbid associates, all being due to a central cause. As to the local sweatings and pigmentations, these and other symptoms were far from being constant; pigmentation especially was common in elderly people who were not affected with osteo-arthritis. The influenza epidemic appeared to have materially increased the amount of

osteo-arthritis, and in cases where the disease had already commenced it had been made much worse.

Dr. SPENDER, in reply, said that the material in Bath for the clinical study of rheumatoid arthritis was enormous, and almost bewildering in its variety. He had ventured to bring some of it before the Society that evening, in order to learn from the criticism and to be instructed by the judgment of the members. He must still maintain that the special symptoms which he had described were not chance accompaniments of the disease, but connoting signs in the sense that they demonstrated what the disease was. It was not pretended that pain or quick pulse or pigmentary deposition *made* a case of rheumatoid arthritis. The arthritis and the muscular atrophy were the essential points; but the nature of the atrophy and the arthritis might be proved, if otherwise doubtful, by the collateral phenomena. One or more of these had been observed in such a large proportion of cases that there must be some pathological connection between them. And only on a neural hypothesis could such a connection be established.

A CASE OF INFECTIVE ENDOCARDITIS OF RIGHT SIDE, WITH PNEUMONIA AND CEREBRO-SPINAL MENINGITIS.

By SIR DYCE DUCKWORTH, M.D., LL.D.

HENRY C —, aged 28, a potman, was admitted into Matthew Ward, in St. Bartholomew's Hospital, under my care, on June 25th, 1891, complaining of pain in his chest. On the 21st June he shivered, and suffered from shortness of breath and cough. On June 22nd he was worse, with pain shooting through the chest to his shoulders, and took to bed. The pains increased, and another shivering occurred; dyspnœa continued severely, and there was some delirium.

The family history was unimportant. There was no antecedent history of any important illness, and none of rheumatic fever. He had been an intemperate man.

On admission he was found to be fairly well-nourished. The face was flushed and dusky, and the large veins turgid. There was some cough and dyspnœa, the former painful. Skin moist, alæ nasi dilating, conjunctivæ icteric. No herpes detected. Tongue thickly furred posteriorly. Great pain complained of on the right side of the chest. The temperature was 102°, respirations, short and catching, 48, and the pulse 140, soft. Appetite lost.

The chest expanded feebly. Percussion-note fairly good under

each clavicle, dulness in the right axilla. Over the right front double friction audible, and faint bronchial breathing, especially with expiration. The left front was also dull at level of sixth rib, and in the axilla, with friction, and doubtful pleuro-pericardial friction audible. Behind, both backs were found dull to percussion to the inferior angles of scapulæ, and an impaired note as high as the spines of the scapulæ. Bronchial breathing, friction, and bronchophony audible, with very few crepitations. Sputa tenacious, and of greengage plum colour.

The heart's apex was just inside the left nipple-line. Sounds clear. No increase of cardiac dulness observed.

The abdomen was natural, save for some indication of scabies on the integument.

The extremities were natural, but rather dusky. Moisture in the palms of the hands.

The bowels were loose, five motions were passed soon after admission. The urine was free from albumen.

June 26th.—Delirious during previous night, but not noisy; took nourishment well. Temperature rose to 102.4° . Ordered a mixture with digitalis, nux vomica, nitrous ether, and chloroform water. Poultices to the chest. Milk, beef-essence, 6 ounces of brandy, and later on 2 ounces of the *mistura spiritûs vini Gallici* every six hours. Thick rusty sputa were expectorated. The pulse rose to 144, and was occasionally dicrotous. Carphology noticed.

June 27th.—Had a fairly good night, with less delirium. At 7 A.M. the temperature fell to 98.6° . Physical signs in the chest as before, with redux crepitation on the right side. Pulse dicrotous. Morphine and ether draught given each night.

June 29th.—Temperature 100.6° , after a fairly good night. Pulse 128, respirations 36. Quinine was added to the medicine yesterday, and minced mutton and jelly were well taken.

June 30th.—General condition rather improved. No longer delirious. Less dusky. Many more moist sounds in lungs. Bronchial breathing at left apex behind. Expectorating more freely. Temperature 101° . Pulse 142. Respirations 40. Tongue rather dry. Urine free from albumen.

July 2nd.—Improvement not maintained. Is again delirious, and dyspnœa is urgent. More dusky, and is sweating. Temperature 102.6° . Hypodermic injections of 3 minims of liquor strychninæ employed, and ether draught given occasionally.

July 3rd.—More drowsy and restless. Continued to take nourishment well.

July 4th.—It was noted that there was less bronchial breathing at the apex of the left lung, and that there were large crepitations at both pulmonary bases with less dulness on percussion. The sounds of the heart were noted as "clear." The temperature reached 103°, the highest recorded during the illness, this being the fourteenth day.

July 5th.—There being much dyspnœa and increasing cyanosis, inhalation of oxygen gas was tried. No benefit was gained. Some convulsive seizures occurred, and death ensued on July 6th.

Post-mortem Examination, 7th July, 1891.—Infective endocarditis, right side; consolidation of both lungs; abscess in one lung; cerebro-spinal meningitis.

Head.—Bones *nil*. Effusion of lymph at base of brain, extending up Sylvian fissures, between the two halves of the cerebrum, and down the cord. Sero-purulent effusion in ventricles which are somewhat dilated. Brain substance soft.

Pleuræ and Lungs.—A few adhesions in both pleuræ. General engorgement of lungs. Right lung, irregular consolidation at base, and towards centre of it an abscess, size of a pigeon's egg. Left lung: some red hepatization at base.

Heart.—No pericarditis. No hypertrophy. On the auricular surface of tricuspid valve a large irregular racemose growth, equal in size to three or four small grapes, soft and friable, and of dark grey colour. Undergoing putrefactive changes. (Sent to museum.)

Abdomen.—Some adhesions about spleen.

Liver.—75 ounces, pale, smooth, fatty.

Spleen.—Very large and soft. No infarcts.

Kidneys.—Large and engorged.

Ureters and Bladder.—*Nil*.

The foregoing case was noteworthy in several particulars. In the first place, a diagnosis of its essential nature was not made. The phenomena agreed sufficiently with those recognised as constituting an attack of double pleuro-pneumonia in an intemperate young man.

It is especially to be noted that nothing pointed to cardiac complication, there being neither physical signs nor symptoms of any such trouble.

There was nothing extraordinary in the occurrence of delirium

and a marked degree of adynamia in the case of a drunkard struck down with double pneumonia. Diarrhœa and carphology in such a case were also not specially noteworthy indications.

It may further be noted that the rigors were confined to the earlier stage or onset of the illness. Certainly, none occurred after admission to the hospital on the fifth day of the disease, and hence no special indications of progressive septicæmia were manifested. The sweating was not undue or remarkable. The highest temperature recorded was on the fourteenth day, 103°. There was something like a critical fall of temperature on the seventh morning, but the pyrexia went on its course forthwith, and a fatal issue was reached on the sixteenth day.

In cases of infective endocarditis we can only look for direct effects in the line of the circulation, and as the mischief occurred here on the auricular surface of the tricuspid valve, the effects were mainly manifested in the lungs, and this in the shape of a septic pneumonia.

The cerebral symptoms were not specially noteworthy, and not till the last day of the illness did any convulsive condition occur. By that time there was seemingly a sufficient degree of carbonæmia to explain these.

I suppose it must be admitted that cases of this kind may occasionally escape recognition at the bedside.

There was no history of special exposure to induce pneumonia, and pain in the chest appears to have preceded the first rigor. Herpes was absent from the lips, as is so often the case in septic, as distinguished from herpetic, pneumonia arising from chill. This is not an invariable rule, and it may be met with in gouty pneumonia. Albeit, I think more favourably of cases of pneumonia presenting herpes, and the more of it the better.

One is reminded by this case of those examples of pneumonia which are followed by meningitis, and in which diplococci are met with both in the lungs and in the meningeal effusion.

The infective mass in the heart was already putrid when first seen, and no worthy examination of it was possible in respect of specific organisms.

The facts of this case appear to indicate that, although infective endocarditis may arise on one or other side of the heart, yet the general symptoms and the physical signs may be expected to vary very materially in the two cases, and in the hope of eliciting more

knowledge on the whole subject, I venture to submit this example of the disease to the criticism of the Society.

Dr. F. L. BENHAM said that the case just related was obviously one of pyæmia, and that the symptoms described were therefore due to infection by the absorption of poisonous matter from a purulent focus somewhere in the body; but in the description of the case, no mention of such an infective focus was made. He would like to ask whether a thorough and careful search was made for such a cause. He strongly suspected from the presence of meningitis (which was otherwise not accounted for), that the origin was disease of the temporal bone from chronic ear-mischief; hence thrombosis had occurred in the sinuses at the base of the skull, and the disintegration of these thrombi had caused putrid material to be carried into the veins, setting up meningitis locally, and also infecting the heart and lungs.

Mr. SHEILD had seen a number of cases of acute necrosis, which were often mysterious in their onset, being mistaken for rheumatism because of the joint effusion and pericarditis. The condition of the heart was probably always secondary to that of the bone. The occurrence of hæmaturia might be expected owing to renal embolism. Gangrene of the limbs might result in cases of pyæmic thrombi from valvular disease of the heart.

Dr. A. GASTER mentioned a case which had come under his notice, having the same characteristic features as that treated by Sir Dyce Duckworth. A patient, who had received an injury on the right knee, entered the surgical wards of the Filantropia Hospital, at Bucharest. The joint was tapped, and pus was discharged from it. In the course of treatment, signs of pneumonia supervened, with delirium, high fever, &c. At the heart only a very faint systolic murmur could be heard over the xyphoid appendix, but no organic disease of the heart was thought of. At the *post-mortem* some exudation and fibrinous deposits like pus were found over the cerebellum. In the right lung several abscesses, varying in size. In the left lung several nodules of catarrhal pneumonia. In the heart, between the anterior and internal valve of the tricuspid, a vegetation presenting a ragged ulceration of $1\frac{1}{2}$ cm. diameter, which penetrated the thickness of the interventricular septum, but the endocardium of the left ventricle and the valve remaining healthy. Professor Dr. Babes, who investigated the case bacteriologically, came to the following conclusion:—"It was a case of endocarditis, with an exceptional localisation, caused by an unknown bacterium, in every probability belonging to the *Proteus* species (very pathogenic to rabbits and white mice), associated with the *Streptococcus* and the *Staphylococcus aureus*. The latter was found during life only in the urine, whilst after death nowhere else than in the kidneys."

N.B.—The notes of the case with bacteriological investigations were published in the 'Annales de l'Institut de Pathologie et Bactériologie de Bucharest,' par Victor Babes, Part I, 1 année, page 382, under the heading "Association Bactérienne dans l'Endocardité," par V. Babes.

Dr. CHAPLIN said, a few months ago he exhibited at the Pathological Society a heart, in which the pulmonary valves and the pulmonary artery were affected with acute ulcerative endocarditis. There was a vegetation on the pulmonary valves, and this vegetation was connected with a thrombus, which completely filled the pulmonary artery, and was adherent to the wall of the vessel; on separating the thrombus, the wall of the

vessel could be seen to be eroded. There was a small vegetation on the mitral and aortic valves, but the tricuspid valves were free. In the lungs there were infarcts and small patches of red hepatization. This case, like the one under discussion, was a case of right-sided ulcerative endocarditis, and, like it, attention was for a time wholly directed to the lungs. Soon, however, a double murmur appeared over the pulmonary artery, and this, in conjunction with a hectic temperature of 104° every evening, and a petechial rash on the backs of the hands and on the face, enabled one to assume with tolerable accuracy that ulcerative endocarditis was present.

Sir DYCE DUCKWORTH expressed his thanks for the criticism offered on his paper. In reply to Dr. Benham, he was not able to state whether the condition of the middle ears had been specially investigated at the autopsy.* Clinically there was nothing in the case pointing to disease in these organs. There was nothing specially remarkable in the occurrence of infective endocarditis, even of the right side of the heart, but the point which he urged in this case was the entire absence of all symptoms pointing to the heart as a source of the septic pneumonia and cerebro-spinal meningitis which occurred. No special indications of septicæmia were present after admission to the hospital. The heart-sounds were clear, and all the features of the case pointed to a pleuro-pneumonia of adynamic character in a man who was habitually intemperate. The case was essentially one of general pyæmia, and came into relation with those forms of that disorder which sometimes started in the bones, as in acute necrosis, where ulcerative endocarditis occasionally supervened, together with other pyæmic deposits.

May 2nd, 1892.

THE ANNUAL ORATION—SEX IN EDUCATION.

By SIR JAMES CRICHTON-BROWNE, LL.D., M.D., F.R.S.

MR. PRESIDENT AND GENTLEMEN,—When that eccentric father of a family of geniuses, the late Rev. Mr. Brontë, desiring that his children should speak freely and without timidity, put them behind a mask and questioned them on various subjects, he was told by his son Branwell, then 7 years old, in answer to one of his interrogations, that the best way of knowing the difference between the intellects of men and women is by considering the differences between them, as to their bodies. That deliverance of the precocious boy seemed to his father at the time a wise saying, worthy of being recorded, and I daresay it seems the same to us as medical men to-day, but it is incontestable that there are now

* Subsequent examination of the clinical notes and of those taken at the autopsy, show that no special inquiry was indicated or made on this point.

large numbers of cultivated persons to whom it must sound as foolishness, and a mere infantile echo of a barbarous prejudice. Their reply to Mr. Brontë's question would be that it is impossible to distinguish between the intellects of men and women, as there is no difference between them. Bodily differences cannot be overlooked; they are still obtrusive and are admitted; certain emotional differences are, perhaps, conceded, but as regards intellect, we are told that in its pure atmosphere all gross sexual characteristics disappear. The intellect of woman, it is maintained, except in so far as it has been enfeebled by long ages of subjection, is as good as that of men, if not better. And even where this extreme view is not theoretically held, it is often practically acted on, for those who admit that there are certain differences between the male and female intellect still often advocate the co-equal and co-ordinate employment of men and women in all intellectual exercises and pursuits. There is a growing tendency around us to ignore intellectual distinctions between the sexes, to assimilate the education of girls to that of boys, to throw men and women into industrial competition in every walk of life, and to make them compeers in social intercourse and political privileges. And as to my thinking, this tendency is unphysiological, and likely, if indulged, to lead to some unfortunate results, I seize the opportunity, which, by your kindness, I enjoy this evening, to vindicate the wisdom of Master Branwell Brontë, to insist that there are differences between the intellects of men and women, and that these are best understood by a study of the differences in their bodies, and to suggest that forgetfulness of these differences is already doing injury in one department of education, I mean the high school education of girls.

Now, to catalogue, in the briefest way, the bodily differences between men and women which underlie their intellectual disparities would be to exhaust the time at my disposal. They are universal and intimate, and involve every organ and tissue. They extend from cuticular appendages to the marrow of the bones, from the crown of the head—for, according to Broca, the female cranium is less elevated than that of the male—to the sole of the foot—for, according to Delaunay, woman has a plantar arch, flatter than that of man, which, perhaps, accounts for her partiality for high-heeled boots. I shall not attempt such an extensive anatomical survey. My present purpose will be served by directing your atten-

tion to certain sexual differences in one bodily organ—the brain—differences which have been greatly lost sight of, which much require further investigation, and which are of peculiar significance in connection with intellectual manifestations.

But before I do so, I wish to say one word as to the origin of these sexual differences, which are so all-pervading. I am not going to attack the problem of the determination of sex—of which, it is said, 500 solutions have been attempted, and which still, as far as I can judge, defies scientific analysis; but I desire to remind you that the radical explanation of sex is to be sought in what Michael Foster has called “the protoplasmic movement,” that is to say, in the integrative and disintegrative changes of living matter. It is the preponderance, on the one hand, of the assimilative and synthetic processes by which protoplasm appropriates dead matter, and transforms it into more complex and unstable compounds, and, on the other hand, of the disruptive and analytic processes by which protoplasm breaks down its own substance into simple and stable constituents, to be excreted as waste products, that determines the specialisation of the cell. The synthetic and assimilative processes are summed up under anabolism, and from predominant anabolism the female emerges. The disruptive and analytical processes are summed up under katabolism, and from predominant katabolism the male emerges. In predominant anabolism there is storing up of nutritive material, with accumulation of potential energy and great passivity; in predominant katabolism there is waste of nutritive material with expenditure of kinetic energy and great activity. If we suppose that hermaphroditism was the primitive state among unicellular organisms, we can perceive how unisexualism was evolved out of it through the phases of metabolism. Amongst the Protozoa in which the beginnings of dimorphism can be discerned, and which correspond with the reproductive cells of higher animals, we have in amoeboid organisms, which are large, inert, passive and spheroidal—in which anabolism preponderates—a forecast of the quiescent ovum, and in infusorian organisms, which are small, active, ciliated or flagellate, we have a forecast of the mobile spermatozoon. By virtue of predominant anabolism female cells are more nutritive, by virtue of predominant katabolism, male cells are more variable. And the bodies which result from the union of these cells are like unto them. The primary physio-

logical differences in the reproductive cells are repeated in the individuals which result from their union, male and female, and permeate these individuals, creating in the one a tendency to a rapid breaking down of complex molecules and a corresponding activity or expenditure of energy, and in the other a tendency to the building up of molecules with corresponding passivity or storage of reserve energy ; and it is these tendencies that are the architects of men and women, and that, in every detail of their structure and functions, from the foundations of bone to the finials of fancy, work out their archetypal ideals.

Anabolic and katabolic processes are manifold, vary in their relative ascendancy in different individuals, and are influenced by environment, so that, in tracing their operation through the animal kingdom, qualifications and explanations are from time to time needful ; but subject to these, it is everywhere obvious that the female is the outcome and expression of predominant anabolism, and the male of predominant katabolism. A study of the organic and functional, primary and secondary sexual characteristics, of the normal development of the tissues and of their pathological modifications makes this evident, and a study of the emotional and intellectual characteristics of men and women leads to the same conclusion. Man is more wilful, enterprising, passionate, and energetic, that is to say, more katabolic, in the mental sphere, while woman is more receptive, tranquil, affectionate, and constant, that is to say, more anabolic in the mental sphere. His restless habits give man a wide range of experience, and so amplify his intelligence, but her narrower existence concentrates her powers, and so quickens her perceptions. Sudden in impulse, brave and independent, fickle, and eager after novelty, man is more original, while woman, patient, trustful, compassionate, and timid, excels in rapid intuition. Man ranges afar into the past and future. Woman is content to dwell in present bliss ; man is experimental : woman conservative. He has grasp and scientific insight : she subtlety and common sense. Her intellect is chiefly integrative or anabolic, his is analytic or katabolic.

Well, this brings me back to the point from which I started on this outline of the origin of sex, namely, the brain-differences between men and women ; for, of course, differences in intellect imply cerebral differences, and it is of importance to ascertain what these are.

And first, amongst cerebral differences between the sexes, I would refer to mass and weight, qualities with which one almost insensibly associates power and strength. Now it is a matter of common observation that women have smaller heads than men, and it is a matter of scientific observation that in all peoples and races without exception, the absolute weight of the entire brain is, on the average, greater in men than in women, though, of course, individual women do sometimes possess larger and heavier brains than individual men. But it is also a matter of scientific observation that there is a correlation between brain weight and stature, and, laying hold of this fact, the advocates of woman's rights and might have argued that the deficiency in her brain weight, when compared with that of man, is no more than is to be accounted for by her fewer inches. But this position is quite untenable. I do not know a trustworthy standard of the brain weights of healthy natives of this country to which to appeal, but I can submit to you a table showing the results of the weighing of the brains of a large number of insane persons, all English, Scotch, or Irish, but mostly English, which, in this relation, is absolutely reliable.

TABLE I.—*Brain Weight.*

Sexes.	Average weight of brain.		Average height.		Excess of male over female brain weight.		Excess of male brain weight after allowance for height.	
	Grammes.	Ounces.	Metres.	Feet. Inches.	Grammes.	Ounces.	Grammes.	Ounces.
945 males ..	1350·54	47·64	1·702	5 7	127·68	4·50	29·71	1·05
655 females	1222·86	43·14	1·575	5 2	—	—	—	—

In this table are summed up the brain weights of 1,600 persons, 945 males, and 655 females, ranging from 10 to 80 years of age, the weighing of the brain having been in each case conducted by myself or under my own supervision. You will observe that the brains of males exceeded those of females in weight by 127·68 grammes, or 4·50 ounces on the average, and that after

allowing for differences of stature, taking 5 feet 7 inches as the average male height, and 5 feet 2 inches as the average female height, there is still an excess of brain weight of 29·71 grammes, or 1·05 ounce in favour of the male. This is a very substantial difference, and, if we recollect that the brain of the ant, of the mental powers of which Sir John Lubbock has spoken in such complimentary terms, declaring that they differ from those of man not so much in kind as in degree, is of the size of a pin's point, we shall realise that an extra ounce of brain matter within the human cranium may imply an enormous mental difference. But it is certain that the actual difference in brain weight between healthy English men and English women is much more than 1 ounce. My table, as I have told you, deals with lunatics in asylums, and amongst them organic diseases of the brain, involving loss of substance, are at least twice more frequent in men than they are in women. Women are oftener attacked by insanity, but men oftener die of it. General paralysis of the insane, atrophy, and softening of the brain, senile dementia, and other diseases, causing wasting of the convolutions, are far more fatal to men than to women, who, when they die in asylums, succumb most frequently to bodily diseases which do not seriously interfere with the nutrition of the brain. And so it comes about that of the brains weighed in the *post-mortem* theatre of an asylum, those of men are on the average much more water-logged and reduced below their normal bulk than those of women. The effect of organic diseases, and the atrophic changes they induce in lowering, in my tables, what is the normal difference between the male and female brain, may be statistically demonstrated from these tables themselves. It is from 30 to 50 years of age that the mortality from general paralysis which destroys four men to one woman, and from other fatal forms of organic brain disease, preferentially attacking men (except, of course, senile dementia), almost exclusively occurs, and it is in this section of life that the sex difference in brain weight in my tables falls to the lowest point at the very time when under normal circumstances it ought to be greatest. The average excess of weight in the male as compared with the female brain from 30 to 50 years of age in my tables was 123·5 grammes, or 4·37 ounces, while from 20 to 30 years, when deaths from organic brain disease are rare, it was 169·9 grammes, or 6·01 ounces. All available evidence points to the conclusion that the male brain exceeds the female

brain in weight in this country to an even greater degree than has been hitherto believed. And that the smaller size of the female brain is a fundamental sexual distinction, and is not to be accounted for by the hypothesis that environment, educational advantages, and habits of life, acting through a long series of generations, have stimulated the growth of the cerebrum in one sex more than in the other, is made clear by the fact that the same differences in brain weight between men and women have been found in savage races. And not only is the male brain heavier than that of the female, but it has a wider range of variation in weight. The very big brains and the very small brains are encountered just as are geniuses and idiots, giants and dwarfs, more frequently amongst men than amongst women.

I have said that an extra ounce of brain matter within the cranium would involve an enormous mental difference. It would do this were it generally and equally distributed; and it would do so in a still more striking manner were it localised in a certain region of the cerebrum. And there are grounds for believing that there is a difference in the balance of parts in male and female brains respectively, and this difference I adduce as the second sexual distinction between them. Broca, no mean authority, has declared that the occipital lobes are more voluminous in the female than in the male, and my own observations, published in 'Brain,' in 1880, confirm, as far as they go, his conclusion, and show that while the frontal lobes are equally developed in both sexes, the parietal lobes, corresponding roughly with the motor area of Ferrier, are larger in the male than in the female, and the occipital lobes, certainly sensory in their functions, are larger in the female than the male.

The third brain difference between the sexes to which I would allude is one of convolutional arrangement, which cannot yet be accurately defined, but which the examination of a series of photographs of brains of men and women which have been stripped of their membranes pretty clearly reveals. The brains of women, like their bodies generally, are upon the whole more symmetrical than those of men. The difference which I have found in the weight of the hemisphere points to this conclusion, for in males the right hemisphere exceeds the left in weight by 3·7 grammes, and in females by only 2·1, but pathological considerations as to the regional distribution of wasting in organic diseases forbid me to attach too

much importance to this observation, and I prefer to trust to mere ocular inspection, which will, I think, bring home to anyone who diligently uses it, the superior symmetry of the female brain, due to its comparative poverty in secondary gyri.

It is in the internal structure of the brain, in the depth and arrangement of its grey matter, in the size, form, and connexions of the cortical cells, in different areas, that the most essential differences between the male and female brain in all probability reside, but the internal structure of the brain in this relation is as yet uninvestigated. I may mention to you, however, one fact which suggests some difference in the constitution of the grey matter of the brain in the two. During a very laborious inquiry into the specific gravity of the grey matter of every gyrus of the brain, which I carried out some years ago, I succeeded in obtaining three standard brains of perfectly healthy adults, two men and one woman, killed in accidents, and in these I found that while the specific gravity of the medullary substance of the brain was alike in all, in each region, namely 1044, that of the grey matter varied remarkably. To take the frontal convolutions as an example, in the one male the grey matter of these had a specific gravity of 1037, in the other of 1036, but in the female its specific gravity was only 1034. And not only in the frontal region, but in every convolution the specific gravity of the grey matter was lower in the female than in the male. I do not lay any great stress on the observation of a single case, but in several cases of death during acute insanity, which I examined, in which there had not been time for the establishment of degenerative changes, the specific gravity of the grey matter in every lobe of the brain was invariably lower in the female than in the male, while the specific gravity of the white matter always closely corresponded in the two. In all degenerative diseases of the brain, and especially in senile decay, there is a reduction in the specific gravity of the grey matter, which sometimes falls as low as 1028 in the frontal region: and the fact, if it be a fact, that the grey matter in the female brain is of less density than that in the male brain must mean that it is a less highly nourished and developed, and a more watery tissue.

And there is still another brain difference between men and women which I must submit to you, and that a very momentous one, namely, vascular supply. During the last four years Dr.

Sydney Martin and I have, as opportunity has offered, carried on an inquiry as to the size of the great arteries that supply the brain. The details of our observations, which have been conducted by Dr. Sydney Martin with scrupulous care, and by new methods, insuring, I believe, great accuracy of measurement, will be communicated to the Royal Society when they are complete, but I may mention now one or two of the results at which we have arrived. We have found that the diameters of the internal carotid and vertebral arteries, taken together, are slightly greater in the male than in the female. Their combined diameter is 8.2 mm. in the male and 8.0 mm. in the female, but when the difference in size of the male and female brain is taken into account, it is found that in proportion to brain weight their diameter is greater in the female than in the male, and so it appears that upon the whole the female brain receives a larger supply of blood in proportion to its mass than does the male brain, but of course it is to be remembered that the blood going to the female brain is somewhat poorer in quality than that going to the male brain, and contains only 4,500,000 corpuscles to the cubic millimetre, instead of 5,000,000 in the case of the male. But Dr. Sydney Martin and I have found further, that the internal carotid and vertebral arteries when measured separately display a marked difference in calibre in male and female brains respectively. In 10 brains of male adults free from brain disease, and ranging from 25 to 36 years of age, the internal carotid arteries had an average diameter of 2.8 mm. on the right side and 2.75 mm. on the left side, while the vertebral arteries had an average diameter of 2.2 mm. on the right side and 1.875 mm. on the left side, whereas in 10 brains of female adults free from brain disease, and ranging from 25 to 43 years of age, the internal carotid arteries had an average diameter of 2.6 mm. on both sides, while the vertebral arteries had an average diameter of 2.3 mm. on the right side and of 2.075 mm. on the left side. It thus appears that the distribution of the blood in the male and female brain respectively varies to a considerable extent. The internal carotid arteries, with their great branches, the anterior and middle cerebral arteries, supplying the supra-orbital convolutions and island of Reil, the gyrus fornicatus, the Rolandic area, the angular gyrus, and the first temporo-sphenoidal lobule, are much larger, both absolutely and relatively, in the male than in the female brain; but the vertebral arteries

which supply the occipital and temporo-sphenoidal lobules are larger in the female than in the male brain, and the basilar artery, which is practically a continuation of the vertebrals, is also larger in the female brain, where its average diameter is 2.8 mm., than in the male brain, where its average diameter is 2.675 mm. It might be thought that the free anastomosis provided by the circle of Willis renders comparatively unimportant differences of calibre in the internal carotid and vertebral arteries, and must equalise the blood currents to the different regions of the cerebrum; but the fact is, that the posterior communicating arteries, which when dilated after the occurrence of any pathological obstruction on the cardiac side of the circle of Willis, maintain the circulation in the brain in tolerable integrity, are incapable while normal, by their calibre and position, of adjusting the balance between the direct currents of the carotid and vertebral arteries; and it is certain that the result of the difference in the diameter of these in the two sexes which I have recorded is this, that the anterior region of the brain is comparatively more copiously irrigated with blood in men and the posterior region in women. And vascular supply is in some degree a measure of functional activity, the flow of blood to an organ or part having always a relation to its working power. But the region of the brain, which in men is most richly flushed with blood, is that which is concerned, we have reason to believe, in volition, cognitions, and ideo-motor processes; while the region which in women is most vascular is that which is mainly concerned in sensory functions, and we thus see that there is a relation between the size of the cerebral arteries and what observation has taught us as to the intellectual and emotional differences of the sexes.

The structural differences between the male and female brain, which I have chiefly referred to, justify the conclusion that they are organs broadly distinguished from each other, and that they have to some extent different kinds of work to do. All through life, the male brain differs from the female in capacities, aptitudes, and powers. Differences early assert themselves. Thackeray has said that little girls make love in the nursery, and practise the arts of coquetry on the page boy who brings the coals upstairs, and as for the page boy, it is certain that his pugnacious propensities are already fully developed, and have brought him into conflict with his brother buttons. And differences are most patent of all

in the prime of life, when man, "for contemplation and for valour formed," by "his fair large front and eye sublime," declares "absolute rule," and when woman, "grace in her steps, Heaven in her eye, in every gesture dignity and love," stands conspicuous for "softness, and sweet attractive grace." And differences subsist to the last. The aged spinster, left in "maiden meditation, fancy free," lavishes her altruistic emotions on cat, poodle, or parrot, and the hoary veteran, fidgetty and irascible, concentrates his egotistic attention on his own liver. And these differences in brain structure and function, which at every stage of existence separate the sexes, have, as I shall presently show, a special pathological significance at the period when sexual divergence is taking place most rapidly, and when education is being pushed forward with most vigour. Education, from first to last, can only be safely conducted in the light of cerebral physiology; but, unfortunately, those charged with the conduct of education too often dispense with that light, or regard it as misleading. It is to point out to you the risks that are run by dispensing with that light at a particularly dark and tortuous part of the educational highway that I am here this evening. I am going to notify, for warning and guidance, some of the perils to health which seem to me to attend the high school education of girls, which is now so popular.

I have no wholesale indictment to bring against high schools for girls. They have done good service to sound education, have widely diffused its benefits, have supplanted second-rate boarding and venture schools which were hot-beds of namby-pambyism, and have opened up to girls interests, and helpful attainments, which were formerly denied them, thus saving some of them from a vapid and weary existence. But, at the same time, it seems to me, that these schools have serious drawbacks attending on them, and that their work is apt to involve very grave dangers to health—dangers immediate and prospective—which have not yet been sufficiently appreciated.

Even from an educational point of view, the work done by high schools for girls is not all pure gain. Their eulogists would have us believe that they have led forth great hosts of girls from a wilderness of ignorance and ineptitude into a land flowing with wit and learning. But that is sheer nonsense. What they have done is to conduct them from the unkept meadows of natural growth into the trim gardens of artificial culture. Excellent are

orchids and camellias in their way, but do not let us forget the buttercups and daisies. Before the high school era dawned, girls lived and learned and reasoned in a way, and in introducing them to the higher erudition, these schools have withdrawn them to a large extent from homely household occupations, which were not without their educational value, and have substituted the dogmatic teaching of the hireling for the precept and example of the mother. So much is this the case, and so impossible is it for growing girls, exhausted by five or six hours of school work and private study daily, to make themselves acquainted with domestic economy, that it seems to me essential that high schools, if they are faithfully to prepare their pupils to become efficient wives and mothers, should add housewifery in all its branches to their present curriculum. Two years ago I met in the country a high school girl who was reading Lucretius for her recreation, but she failed lamentably in the task I prescribed to her, that of boiling a potato. Now I am sure much more of the happiness and wholesomeness of life hinges on the boiling of potatoes, than on the interpretation of Lucretius and his dark and doubtful sayings. And not only do high schools for girls deprive their pupils to a considerable extent of home-lore and practice in the simple but captivating arts of the kitchen and the still-room, but they tend to induce in them sameness and narrowness of intellect. These schools cannot vary or adapt their teaching to individual tastes and talents, but have one keynote for all, and so sacrifice to clearness of utterance many delicate inflections and cadences of faculty. They tend to make education mechanical. Then they so absorb the energies of their pupils and so persuade them of the paramount importance of their school work that these pupils have neither inclination nor strength to travel beyond their compulsory studies. With brains like wrung sponges, and well assured that there is nothing worth knowing beyond the attainments of the sixth form, high school girls rarely leave the groove in which they find themselves. It will not be denied that home-reared girls read much more widely than do high school girls. "They browse unconfined," to quote Mrs. Gaskell, "on the wholesome pasturage of English literature," while high school girls are stall-fed on condensed primers, and the result is that there are often a breezy freshness and interesting diversity about these home-reared girls that contrast not unfavourably with the dry precision and

monotonous uniformity of their more systematically educated sisters.

But it is not about the educational advantages or drawbacks of high schools for girls that I wish to speak, but about the dangers to health which lurk in their aims and methods—dangers very real, very serious, very imminent—all arising out of forgetfulness of sexual differentiation, and out of the futile attempt to educate boys and girls on exactly the same lines.

Now I do not hesitate to affirm that over-pressure is rampant in high schools for girls in this country to-day. Of course there are high schools and high schools. Much depends on the head mistress. If she is judicious and sympathetic, over-pressure is reduced to a minimum. If she is hard and keen, it is raised to a maximum. But whatever the disposition of the head mistress may be, over-pressure exists and is promoted by directors, jealous of the honour of their schools, and not adverse to dividends; by parents, anxious about their daughters' prospects in life, earnest to obtain the best possible return for fees paid, or not unwilling to hide their own dulness under the lustre of having clever children; and by the girls themselves, who, when the spirit of emulation is stirred in them, or the fear of failure conjured up, strain eagerly forward and ignore all warnings and restraints. I willingly admit that in high schools for girls generally there is a sincere desire to avoid over-pressure, and that precautionary measures to prevent it are adopted, and I have no doubt that during the last five or six years there has been considerable mitigation of it, as a consequence of a limit put to the scramble after examinations and certificates. But I am confident that, notwithstanding all precautions and mitigations, over-pressure still prevails in these schools extensively, and sometimes acutely, and that it will prevail as long as they imitate schools for boys, fail to recognise sexual distinctions, and to modify their methods in accordance with these. They are, as I have said, pushed on to over-pressure by certain extraneous forces, but the tendency to it is inherent in their constitution, and will assert itself from time to time with greater or less violence, apart from outside influences, until that constitution is altered. I have no doubt that this statement of mine that over-pressure exists in high schools for girls will be strenuously, perhaps scornfully, denied. These schools have many attractive features, and are doing good work: the girls seen

streaming in and out of them look healthy and happy enough, and those who are content to judge by superficial appearances will pronounce them sound to the core. Stethoscopy and endoscopy, medically applied, are necessary to get at their weak points. Then again there are those who believe that over-pressure is merely a medical myth: that there never was or could be such a thing in the beneficent realm of education, and they will of course dispute my thesis. I read some time ago, a paper by a distinguished authority on educational subjects, Dr. Emily Bryant, in which she argued that it is impossible to over-work girls, their inherent indolence and frivolity being proof against any stimulus that can be applied to them. Well, I would answer Dr. Emily Bryant that it is possible to over-work horses—witness splints, curb, thorough-pin, and back sinew,—and surely girls are not more obdurate than horses; and I would tell her further, that a cloud of competent witnesses can be summoned against her, for while many high school mistresses will doubtless deny that girls are over-worked in the establishment under their care, they will one and all admit that over-work is a contingency against which they have to be constantly on their guard. Some high school mistresses of the more enlightened type frankly admit and deplore the existence of over-pressure even now. One of them said to me not long ago, “Certainly some girls in the upper forms are delicate, owing to too close application to their studies, and it is terrible to contemplate the ruin that might be wrought amongst them were they to be ruthlessly urged on and subjected to competitive excitement.”

For obvious reasons it is difficult to get direct and trustworthy evidence about over-pressure in high schools. These schools are naturally disinclined to permit the exposure of blots on their own escutcheons, and individual cases of over-pressure have to be delicately handled. But thanks to the kindness and magnanimity of the head mistress of one English high school, one too in which special precautions against over-pressure were taken, I obtained, a few years ago, a return, which I regard as of great interest, and which throws some light on the matter. The facts on which this return is founded were got at by questions, put and answered in writing, the answers sent in by each girl being afterwards tested by private cross-examination, by her form-mistress, and in some cases by communications with parents, so that the return is, I believe, absolutely reliable. And it is certainly not a little

TABLE II.—*High School for Girls.*

Form.	No. in form.	Average age.	Which part of your work do you find hardest?	When do you do your home work?	Do you suffer from headaches?	How often do your headaches occur?	At what part of the day do your headaches occur.	Short sighted.	Muscular twitchings.
VI and V ...	17	17	Home work ... 15 Class work ... — Equal ... 2	Afternoon ... 4 Evening... .. 2 Afternoon and evening 11	No ... 5 Yes ... 12	Occasionally ... 7 Frequently ... 3 Habitually ... 7	Morning 2 Afternoon and evening ... 2 No special time ... 7	3	None.
IV ...	25	16	Home work ... 22 Class work ... 1 Equal ... 2	Afternoon ... 5 Evening... .. 4 Afternoon and evening 16	No ... 3 Yes ... 22	Occasionally ... 9 Frequently ... 9 Habitually ... 4	Morning 5 Afternoon and evening ... 14 No special time ... 3	7	None.
Upper III ...	19	15	Home work ... 16 Class work ... 1 Equal ... 2	Afternoon ... 4 Evening... .. 3 Afternoon and evening 12	No ... 4 Yes ... 15	Occasionally ... 9 Frequently ... 5 Habitually ... 1	Morning 5 Afternoon and evening ... 10 No special time ... —	6	None.
Middle III ...	26	15	Home work ... 7 Class work ... 3 Equal ... 16	Morning ... 3 Afternoon ... 6 Evening ... 6 Afternoon and evening 11	No ... 3 Yes ... 22	Occasionally ... 11 Frequently ... 7 Habitually ... 4	Morning 7 Afternoon and evening ... 12 No special time ... 3	4	One.
Lower III ...	24	14	Home work ... 15 Class work ... — Equal ... 9	Afternoon ... 7 Evening... .. 3 Afternoon and evening 14	No ... 8 Yes ... 16	Occasionally ... 11 Frequently ... 3 Habitually ... 2	Morning 3 Afternoon and evening ... 11 No special time ... 3	3	One.
Upper II ...	29	13	Home work ... 20 Class work ... — Equal ... 9	Afternoon ... 15 Evening... .. 4 Afternoon and evening 10	No ... 3 Yes ... 26	Occasionally ... 12 Frequently ... 5 Habitually ... 9	Morning 1 Afternoon and evening ... 11 No special time ... 3	5	None.
II.....	32	12	Home work ... 19 Class work ... 10 Equal ... —	Afternoon ... 19 Evening... .. 3 Afternoon and evening —	No ... 12 Yes ... 17	Occasionally ... 6 Frequently ... 10 Habitually ... 1	Morning 4 Afternoon and evening ... 10 No special time ... 3	5	None.
I	15	10	Preparation in this form is done in the morning. No home work.	Morning ... 15	No ... 8 Yes ... 7	Occasionally ... 6 Frequently ... — Habitually ... 1	Morning — Afternoon and evening ... 6 No special time ... —	4	Two.

remarkable to find from it that out of 187 girls belonging to the upper and middle classes, well fed, and clad and cared for, and ranging from 10 to 17 years of age, as many as 137 complained of headaches, which in 65 instances occurred occasionally, in 48 frequently, and in 24 habitually. The late Sir Richard Owen once said to me, "Children have no business with headaches at all, and if you find that these occur frequently in any school, you may depend on it there is something wrong there." And so I take it there must be something radically wrong in high schools that produce so copious a crop of cephalalgia. That the headaches dealt with in this return were connected with school work is, I think, made probable by the fact that, while in 26 cases they are stated to have occurred in the morning, in as many as 76 they are set down as of most frequent occurrence in the afternoon and evening, when, as you will notice, according to an immense majority of the girls, the hardest part of the day's work falls on an already jaded brain. And there are other significant facts in this return besides those relating to headaches, for you will observe that as many as 37 of the 187 girls were short-sighted, and that 4 of them exhibited choraic movements. And this return represents no exceptional state of things. Inquiries made quite lately satisfy me that a very large proportion of high school girls still suffer from headache, that neuralgia is common amongst them, and that they display multifarious indications of nervous exhaustion. It is no infrequent occurrence, I am told, for the more delicate girls who return after the holidays looking tolerably well, to break down in the middle of the term, to be absent from school for a few days or weeks, to return and struggle on with their work, but finally to shirk the examination at the close of the term. But if we had no evidence of suffering or disability immediately resulting from high school training, as it is now carried on, we, as medical men, should not hesitate to pronounce that training in some respects pernicious, from a consideration of its character alone. Two-thirds of high school girls will attest that the hardest part of their work, preparation, which involves the opening up of new ground, an advance on what has been already learnt, and effort in surmounting obstacles, has to be performed in the evening, when they are already worn out, at the very time when in the cycle of daily life their brains are least capable of exertion. And no inconsiderable number of high school girls will attest that this

arduous work of preparation is often carried on until 10, sometimes even until 11, o'clock at night. Within the last month I came upon two girls attending a London high school, bright, clever, ambitious girls of 15, high in their classes and determined to hold their places, who admitted that, in spite of all restrictions, they were working up till 10 o'clock two or three nights a week, and in one of them I could discern in stunted growth, round shoulders, and intense nervous susceptibility, the effects of this ill-timed diligence in study. A friend of mine in the north of England tells me that he has recently removed his two daughters from a high school and placed them under private tuition, because he found that they were regularly working up till 10 o'clock at night.

The high school authorities will, of course, pronounce such proceedings irregular, and endeavour to shift the responsibility for them on to the shoulders of parents. Their rules as to home work look reasonable enough on paper. This work, they will tell us, should be done during the afternoon, either in school or at home; but girls who have been in school from 9 to 1, using their brains, are not disposed to resume work immediately after their mid-day meal. They need change, fresh air and exercise, which in this climate must, for a great part of the year, be taken in the afternoon, if at all; and many of them continue brain work in the afternoon under the veiled form of accomplishments, such as music and drawing. For these reasons, as well as from the claims of family and social intercourse, it comes about that a large proportion of high school girls postpone their preparation until the evening, when they do it slowly, laboriously, with least benefit and most risk. The work which was calculated to occupy two or three hours is spread over three or four, and perturbs the brain just when it should be subsiding to rest. The time-tables or cards issued to pupils and parents defining with the utmost nicety the number of minutes that are to be devoted to preparation in each particular subject on each particular day, fixing a maximum duration of home work (generally from two to three hours), which is not to be exceeded, without an intimation of the fact to the head mistress, are practically useless, and sometimes worse than useless, for they lead to deception. Not one girl in ten steers by these charts; not one parent in ten reports the transgression of their terms. Girls work at very different rates, and at very different rates in different subjects, and the number of minutes

allowed for Euclid, Latin, German, History, or Science respectively, which may be amply sufficient for a girl of nimble wits, may be altogether inadequate for one of slow comprehension, and hopelessly ill-adjusted for one of special gifts. The relation between the time allowed for home work and the amount of home work allotted is often very difficult to discover, and the highly conscientious girl who tries rigidly to adhere to the instructions given will generally find herself at a disadvantage at school. Parents, for the most part, ignore the school time-tables. They do not always know the exact length of time given by their daughters to home work; it is sometimes secretly prolonged when they supposed it to be finished, and they find that any expostulations as to its amount which they may address to the head mistress are highly distasteful to their daughters, who fear that they will be regarded as stupid, or as a drag on the class, for failing to accomplish their tasks in the prescribed time; or that blame for over-burdening them will be laid on their form-mistress, and of course blame does not conduce to amiability of feeling towards those who have procured its infliction.

The truth seems to be that the checks imposed upon home work are nominal and not real, and that what high school authorities have to do is largely to reduce it in amount, and in the case of young girls to abolish it altogether. The drudgery of education should be done in school with skilled assistance, when the brain is in its prime vigour, not at home unaided, or with only precarious parental help, when the brain is already fatigued. I feel strongly that no girl from 10 to 17 years of age should have any forced brain work to do after 7 P.M., and that a reduction is required in the number of hours that high school girls are now called on to give to brain work. Our brawny colliers clamour to have their bodily labour restricted to eight hours a day. Shall we permit our fragile girls to engage in mental toil for an even longer shift?

I have given prominence to the question of home work in connection with high school education, because I regard it as one of the chief evils of the system, but in connection with the school work there are several topics that are eminently worthy of medical consideration. A criticism of the curriculum and of the subjects taught cannot be undertaken here, but a word must be said about the competition which is still encouraged in some quarters. Nineteenth century girls at the age of puberty cannot stand com-

petition. It is intellectually and morally injurious to them, and disturbs the equilibrium of health. "Nothing," says Ruskin, "is ever done beautifully which is done in rivalry, or nobly which is done in pride;" and nothing, I would add, is ever done safely by girls which is done in emulation. That should be banished from their education, and marks, places, and prizes tabooed. And examinations too, which harass and agitate, should be as much as possible avoided. For girls who have to earn their own living examinations may be necessary trials of fitness, but for the bulk of high school girls they are gratuitous miseries, or mere opportunities of vainglorious display.

I have admitted that in some high schools the authorities are on the alert on the subject of over-pressure, and according to their lights do their best to ward it off. But it is a melancholy fact that some of the expedients resorted to with this purpose tend to aggravate rather than to abate it. One head mistress told me proudly that her practice was, whenever the girls in any form began to get sluggish and drowsy at their work, to close the books and give them ten minutes' hard drill. She thought I was joking when I said that she had much better put them to bed and give them caudle, but I was quite serious, for muscular fatigue is not the remedy for cerebral exhaustion, although it is very commonly believed to be so. Indubitably there is temporary easement in shifting a burden from place to place. A man who has been standing on one leg for some time finds it a relief to change to the other, but the expenditure of nervous energy is going on all the time, and the brain that is well-nigh drained dry needs rest, and will not be replenished by merely altering the channel of outflow. Drill is highly to be commended in its proper place; so are gymnastics; so are games; but they will not create a tolerance of mental over-pressure or counterbalance its evils. It is quietism, not athleticism, that has to be preached in high schools in the first place. I have even my doubts about compulsory hockey and cricket in the country on half holidays. I cannot help thinking that a girl should have just a little scrap of her own life left to her to do with exactly what she likes, and with all respect for physical education—the boom of which is upon us—I have more faith in the life-saving qualities of a good, merry, spontaneous, exuberant romp than in all its ingenious, elegant, and hygienically designed exercises.

The evils resulting from over-pressure in high schools for girls, whether that over-pressure be due to home work, or competition, or examination strain, are brought about through its influence on the cells of the brain, and it is not difficult to understand how these cells are affected. It is unquestionably true of all cells that when they are in any way stimulated in excess of their powers of taking up nutriment, or are deprived of an adequate supply of nutriment, they utilise as food material their own protoplasm, and so induce disordered metabolic processes with subsequent degeneration. In pyrexia, in the presence of which metabolic activity is always increased, we find that the protoplasm of the muscular fibres of the heart and of the cells of the liver passes through a stage of cloudy swelling indicative of increased activity, which merges ultimately into fatty degeneration. And when to the stimulating influence of pyrexia is added that of toxic materials circulating in the blood, as during typhoid fever and diphtheria, we find that generative changes are not confined to the excretory glands and heart, but invade the nerves and muscles generally. Well, it appears to me that the fever of the mind which over-pressure sets up is capable of influencing cell protoplasm in much the same way as ordinary pyrexia. Its effects have not yet been traced out with the same precision. No doubt they differ in degree and distribution, but they are probably much the same in kind. When the cells of the brain are stimulated in excess of their powers of taking up nutriment, as they are during forced mental labour, or when suitable supplies of nutriment are cut off from them, as may be the case during the impairment of digestion which forced mental labour not rarely entails, then the metabolism in these cells is, we may infer, altered, and they degenerate and secondarily induce widespread degenerative changes throughout the system. The group of symptoms which is characteristic of the mental failure which follows upon severe over-pressure, and which may be summed up as acute or apathetic dementia, is almost identical with that seen in cases in which mental failure has followed upon acute specific diseases; and in mental failure of the former kind, not less than in the latter, we have also sometimes dilatation of the heart and changes in the liver, kidneys, and voluntary muscles. And in both kinds of mental failure recovery takes place, if the fatty degeneration has not so far advanced that the active protoplasm of the cells is absorbed, whenever the cells which have had

extra work thrown upon them are allowed absolute rest, and are placed in favourable conditions as regards nutrition.

I have referred to acute or apathetic dementia as the most characteristic of the pronounced mental changes which severe and continued over-pressure acting on a neurotic subject may induce, and the slight mental changes which are similarly induced are most generally akin to it, and are but the buds of the symptoms which it presents full-blown. Thus it is no uncommon event to hear over-worked high school girls complain that their power of acquisition and attention is impaired; that they take far longer to their work than they used to do; that they cannot remember what they have learnt; that they read their lessons without understanding them; that they sometimes lose themselves and forget where they are; and that what they call "queer thoughts" keep coming into their minds; while the observation of these girls at the same time reveals that they are languid and irresolute or unusually irritable. Such slight departures from normal mental states for the most part go no further, but are rectified by the holidays, but now and again they advance into that mild coma which corresponds with apathetic dementia. But besides apathetic dementia there are of course many other mental aberrations to which over-pressure may lead up, the nature of these in each case being determined by the inherited tendencies, antecedents, or environment of the girl. We may have cyclones of mania, or anticyclones of melancholia, hurricanes of morbid impulses, or the settled bad weather of moral perversion. And as regards certain minor mental changes which thus arise, it is noteworthy that they are often concealed by girls who do not comprehend and can scarcely explain them. This is particularly the case with reference to those voluminous mental states described by Dr. Hughlings Jackson, which are sometimes the harbingers of epilepsy.

I cannot pretend to classify or describe the vagaries of nervous disturbance which present themselves to medical observation in girls in these days, and in the production of which over-pressure plays some part, but I would name insomnia, of the commonest occurrence, often of evil import; sopor, or sleep so deep and difficult to break as to be almost cataleptic in nature; neuralgia, chorea, and hysteria. And besides nervous disturbances, there are many ailments and diseases begotten or fostered by over-pressure,

which medical men are familiar with in high school girls in these days. Chief of these is anæmia and general delicacy. Women suffer from anæmia in far larger proportions than men. In the ten years 1881 to 1890, the deaths from anæmia, chlorosis, and leucocythæmia in England and Wales were 33·29 per million amongst men, and 54·83 amongst women. And, as is well known, chlorosis and anæmia show themselves in girls from 10 to 20 years of age more frequently than at any other period of life, and may be induced by mental worry or excitement, which causes a diminished production of blood corpuscles. In his very able investigation into the physical and mental condition of school children, Dr. Francis Warner has satisfied himself that mental stimulus applied to children "does lower their general nutrition." He found that pale and delicate-looking children are proportionately more numerous in schools attended by children of an upper social class, and presumably well fed, than in schools attended by children of the poorer class, presumably not well fed; and the only explanation of this he has to offer is that better class children are subjected to more mental stress than children of a poorer class. A single glance at some high schools, and the complexions of the girls assembled in them, convinces that they are deficient in red corpuscles, however rich they may be in blue stockings.

The anæmia encountered in high school girls is sometimes due to the direct action of mental tension on the blood-forming apparatus, but it is sometimes due also to the action of that tension on the digestive functions. Large numbers of high school girls suffer from loss of appetite; a certain number go to school without breakfast. Worn out, they oversleep themselves, and leave scant time for the morning meal; or, after a night of broken rest, they rise unrefreshed, swallow a cup of tea, the neurotic properties of which they have already discovered, but decline solid food, or merely trifle with it. They labour under a gastric disorder now so common that it might receive a distinctive appellation, and be called *anorexia scholastica*, in which the lessened flow of energy from the exhausted nerve centres retards the functions of all the abdominal viscera. Buns in the forenoon, a regular institution at all high schools now, are very well in their way, but they do not compensate for a lost breakfast, and I hold that no girl who has shirked that meal should be allowed to go to school or engage in brain work. It is an axiom that nutrition comes before education.

I have spoken of some of the immediate effects of over-pressure in high schools—effects which medical men have long recognized, which parents are beginning to perceive—and I wish now to direct your attention to what will probably be its remote effects if it be allowed to go on—effects for which we must wait, but which we have grounds to anticipate with some confidence. The ailments of girlhood which we have ascribed to over-pressure do not always end with the cause that induced them, but are apt to plant themselves permanently in the system they have infested, or to blossom into something worse. The headachy girl is not unlikely to grow into the migrainous and invalid woman. A voluminous mental state may develop into epilepsy, somnambulism may lead up to hysteria, insomnia lays the foundation of insanity, and anæmia at the growth period may entail life-long debility. Over-pressure operates upon the high school girl at a great epoch of her life—at puberty and during the pre- and post-pubertal periods—when momentous changes are taking place in her body and mind, and when a wave of irritability sweeps through her nervous system. The increase of stature and weight of the girl, according to Dr. Bowditch's recent observations, goes on much more rapidly than that of the boy from the 10th to the 14th year, and while her pelvis is expanding, her special organs and mammæ and sebaceous glands developing and becoming more vascular, and her physical energies gathering strength under the governance of new impulses, there exists in the girl special proclivity to disease, and to the assumption of morbid tendencies. This epoch from the 10th to the 17th year is unquestionably the plastic period, it is the golden opportunity for education, but the education that goes on then is not limited to the higher cerebral centres, as teachers fondly imagine, but pervades the tissues. Functional habits are being everywhere formed, and vicious habits are not unlikely to be contracted by certain organs if habits of thought are too assiduously practised. The grand truth to be inculcated in all high school authorities is this, that they have to deal with girls at a period of life when vital resistance is greatly reduced, when the liability to disease is proportionately augmented, and when physiological indiscretions are peculiarly hazardous.

The proof of this can be readily supplied. Let us examine the incidence of zymotic affections upon the sexes. Throughout life, in every quinquennium, the mortality of males from small-pox

exceeds that of females, and that in a very marked degree, except in one quinquennium, from the 10th to the 14th year, when the female exceeds the male mortality, being again but very slightly behind it in the succeeding quinquennium, from the 15th to the 19th year. At all ages the male death-rate from enteric fever exceeds that of females, but the female mortality is very considerably higher from the 3rd to the 20th year of life. In infancy and also in old age the male mortality from diarrhoea and dysentery exceeds the female mortality, but in the child-bearing period, from 15 to 45 years of age, the mortality is distinctly higher amongst females. And even more striking in this connection are the statistics of phthisis than those of zymotic diseases. As you will notice in the accompanying table, phthisis is more fatal to males than females under 5 years of age, but then a change takes place, and from 5 to 10 it is much more fatal to females than to males, while from 10 to 15 it is more than twice as fatal to females as to males. From 15 to 20 phthisis is still much more fatal to females than to males, from 20 to 25 the mortality from it is exactly equal in the two sexes, and from 25 to 30, and at all subsequent ages, the mortality from it is much greater amongst males than amongst females.

TABLE III.—*Phthisis: Mean Annual Death-rate per Million living at successive Age Periods and per "Standard Million" at all Ages during the Ten Years 1881—1890.*

Ages.	Males.	Females.
All ages	1876 ("standard million")	1602 ("standard million")
0—5	527	493
5—10	250	322
10—15	349	712
15—20 . . .	1317	1839
20—25	2350	2355
25—35	3083	2850
35—45	3614	2748
45—55	3574	2085
55—65	2863	1496
65 and upwards	1534	830

Now this distribution of phthisis mortality, especially when viewed in connection with the fact that the reduction in it which is happily going on at all ages has been proportionately less of late years amongst females than amongst males from 5 to 14, is

highly significant. It is not of course suggested that high school operations have had anything to do with this diminished reduction. The total number of girls attending these schools is so small in comparison with the population that no amount of mischief they could do would appreciably affect the Registrar-General's returns. But the facts that there is a special proclivity to phthisis in girls from 5 to 20 years of age, and that girls between these ages are not sharing as fully as boys in the benefits of the preventive measures which we owe to modern sanitation, viewed in connection with the conditions of high school work, suggest very pointedly that one of the remote evils of over-pressure in them will be the propagation of phthisis in those who have been subjected to that over-pressure. The nervous erethism which is characteristic of the growth period of girlhood obviously much increases the liability of the sex to phthisical disease, and as over-pressure aggravates that nervous erethism it must still further increase that liability. The connection between states of the nervous system and pulmonary tuberculosis has not yet been clearly defined, but this much may be taken as established, that nervous depression and exhaustion alike open wide the door for the invasion of the tubercle bacillus. And so it is that the nervous exhaustion due to over-pressure in high schools for girls may be expected to induce a peculiar vulnerability of the pulmonary tissue, which must be intensified by the conditions of work in these schools. The indoor life of girls, their sedentary habits, and the stooping posture in which they pass much of their time, bring the lungs into a state that is favourable to tuberculous infection. The lungs are comparatively immobile, and there is consequent inactivity of the respiratory current in them with a tendency to congestion and catarrh.

If it were our object to secure an abundant harvest of phthisis, I do not know how we could better set about it than by providing for general over-pressure in schools for girls. Keep a large number of town-bred girls in a constant state of nervous tension, so as to abbreviate sleep and impair appetite, deprive them as much as possible of fresh air, insist on their writing and poring over books for prolonged periods, and scatter amongst them a few cases of tubercular disease, and you will inevitably, in the fulness of time, have a rich growth of phthisis. My own experience is necessarily of the most limited description, but I have seen a case

of phthisis in a high school girl, and two cases in high school mistresses, which I was disposed to attribute to high school life, and of this I am sure, that phthisis must be one of the most certain and disastrous of the more remote effects of the over-pressure of girls during their growth period.

But the special proclivity to disease of girls at the growth period, and the influence of the nervous erethism by which they are then visited, may be traced out not only in zymotic diseases and in phthisis, but in nervous diseases themselves. Suicides which spring from causes identical with those that are productive of insanity, and which are an expression of mental disorder, are four times more numerous amongst men than women, and the male death-rate from suicide is much greater than the female at all ages, except, strange to say, from 15 to 20, when the female death-rate rises considerably above the male. The rate of increase of suicides during the last twenty years has been much higher amongst women than amongst men. Chorea, which is at all ages more fatal to females than to males, reaches a rate of mortality amongst females from 10 to 20 years of age that is phenomenal when contrasted with the rates at other ages and in connection

TABLE IV.—*Suicides in England and Wales.*

Mean Annual Death-rate per million living at successive age periods and per "standard million" at all ages during the ten years, 1881—1890.

Ages.	Death-rate per million living.	
	Males.	Females.
All ages	122·3	36·9
0—5	—	—
5—10	0·2	0·1
10—15	4·2	3·0
15—20	29·5	34·1
20—25	66·9	40·1
25—35	118·4	43·3
35—45	204·3	64·1
45—55	315·7	85·3
55—65	428·2	90·5
65 and upwards	454·3	77·8

TABLE V.—*Deaths from Chorea in England and Wales.*

Mean Annual Death-rate per million living at successive age periods and per "standard million" at all ages during the years 1881—1890.

Ages.	Death-rate per million living.	
	Males.	Females.
All ages	2·1	5·7
0—5	1·0	2·0
5—10	4·4	9·5
10—15	4·2	13·6
15—20	2·8	12·9
20—25	0·9	5·6
25—35	0·3	1·4
35—45	0·7	1·3
45—55	1·5	2·2
55—65	3·4	2·8
65 and upwards	4·8	9·1

Years.	Death-rate per million living.	
	Males.	Females.
1870	2·46	4·25
1880	1·75	4·70
1890	1·87	5·40

with the mortality from this disease it is of extreme interest to note that, while the male mortality due to it has declined during the last thirty years, the female mortality has risen, and has risen rapidly, during the last ten years. Chorea is seldom fatal, and the increase of the death-rate from it, amongst girls, must betoken a large increase of the malady in its milder forms. And chorea, be it remembered, is a malady which, perhaps more than any other, may be directly attributed to over-pressure and nervous strain.

I might go on enumerating nervous and other diseases the

mortality from which mounts up from 10 to 20 years of age in girls, but enough has been said to show that there is a dangerous instability of the nervous system at this epoch. And, indeed, the most convincing proof of this instability is to be found, not in the mortality returns, but in the extraordinary prevalence, during this section of female life, of functional nervous disorders, which do not kill, but cripple and perplex, and are often the source of misery long drawn out. Man is, at all ages, more prone than woman to organic diseases of the nervous system, but woman is more prone than man to certain functional disorders, and especially to such functional disorders as display themselves about the period of puberty. Then it is that women succumb to epilepsy, neuralgia, hysteria in all its protean shapes, chorea, migraine, neurasthenia, the milder forms of insanity; then it is that the nervous system is unstable and explosive in a peculiar degree, and especially liable to be influenced, for weal or woe, by the treatment to which it is subjected. Judicious treatment at this time gives it balance, and perhaps steadies it for life. Injudicious treatment—and what treatment can be more injudicious than over-pressure?—either makes it topple over at once, or sets it swaying in a way that must mean final overthrow on the occurrence of any further interference, however slight. Let high school authorities ponder all this.

It has just been mentioned that men are much more liable than women to gross structural lesions of the nervous system, including the various forms of inflammation of the brain and spinal cord and their membranes, paralysis, locomotor ataxia, muscular atrophy, and abscess and tumour of the cerebrum: and there can be little doubt that their greater liability to these lesions is dependent upon, firstly, the innate higher metabolic activity of their nerve centres, and, secondly, their greater exposure during the struggle for life to the causes of nervous disease. Women owe their comparative immunity from organic nervous disease to their anabolic habit, and to the comparatively tranquil and sheltered lives which they have led. But if all this is to be altered, if women are to be made as katabolic as possible, and are to take part in the struggle for life on equal terms with men, then it follows that they will have, to a great extent, to sacrifice the comparative immunity from organic nervous diseases which they have hitherto enjoyed. During the ten years 1881 to 1890, 409 men

per million living, died in England and Wales from the effects of fractures of bones, but only 100 women per million living so died. The explanation of this is clear: the number of men engaged in occupations which expose to accidents, is, to that of women engaged in such occupations, as four to one. But if in our wisdom we ordained that men and women should share equally in manual labour of all sorts, it is indisputable that we should have the mortality from fractures amongst women mounting to as high a point as it has now reached amongst men—nay, the probability is, that it would greatly transcend that point—for the employment of women in all sorts of labour would probably increase the number of accidents, while their anatomical configuration would involve their suffering more severely from their effects. During the ten years 1881 to 1890, 2·69 men per million living, died of tetanus in England and Wales, but only 0·84 women per million living; clearly because, for every one woman, who in her employment has to handle earth, three men have to do so. Expose men and women in an equal degree to inoculation by the tetanus bacillus, and they will in equal degree perish from its attacks. Now, will it be doubted that the same thing holds good with regard to other causes of disease, which operate upon the nervous system, and that the comparative exemption of women from organic nervous disease is to be attributed largely to the fact that they have not been exposed to these causes to anything like the same extent as men? Five men suffer from the motor form of writers' cramp for every one woman who so suffers, and the explanation of this lies on the surface, in the fact that men are employed in writing far more numerously than women. Four men die of general paralysis of the insane for every one woman who so dies; is it not feasible to suppose then that women owe their comparatively small liability to this fell malady to their comparative freedom from the stress and striving of professional and business life, which so often leads up to it in men? To make women katabolic—and that is, I maintain, what high school education tends to do, to throw them into competition with men, and that is, I maintain, what some high school education aims at, is to insure them to a largely increased liability to organic nervous disease. And so over-pressure from 10 to 17 years of age may have, amongst its remote consequences, not only the reproduction in the same or modified forms of the functional nervous disorders which so often manifest

themselves at that period, but a crop of gross nervous degenerations, which have, up to this time, been rarely seen in women. And notwithstanding all Weismann's arguments, I would say, "Woe betide the generation that springs from mothers amongst whom gross nervous degenerations abound!"

The study of the effect of over-pressure, immediate and remote, on the monthly rhythm, and on the fitness and capacity of the woman to reproduce the species and to bear healthy children—health implying both bodily and mental vigour—must be kept for separate examination at some other time. So, also, must a study of the effects of secondary over-pressure, as applied in colleges and halls, to young women at from 18 to 24 years of age. Admirable culture is supplied in these colleges and halls, but they, too, have their risks, notwithstanding that their pupils are all picked lives. The suicide of a pupil who had just undergone examination at one of them was reported two months ago. I must, however, even now express my belief that the University of St. Andrew's in deciding, as it has lately done, to open all its classes in arts, science, and theology, to women as well as men, has taken not a retrograde step—for our ancestors never did anything so foolish—but a downhill step towards confusion and disaster. Its now empty benches may be thronged with pupils, its professors may batten for a time on duplex fees, but the attempt to educate young men and women, not only on the same lines but in the same coaches, cannot but prove injurious to both. "What was decided amongst the pre-historic Protozoa cannot," it has been well said, "be annulled by Act of Parliament," and the essential difference between male and female cannot be obliterated at a sweep of the pen by any *Senatus Academicus*. To essay such work is to fly in the face of evolution. Amongst unicellular organisms, the conjugating cells are exactly alike, and do exactly the same work in the world; but amongst multicellular organisms they are dimorphic, and from that point upwards differentiation in structure and function goes on. With occasional aberrant variations, the sexes diverge from each other, not merely in primary and secondary sexual characteristics, but in functions not directly associated with sex, as we ascend in the animal kingdom, and it is in the human species that sexual distinctions, bodily and mental, are most marked.

And with this divergent differentiation of the sexes has come

more reciprocal dependence and higher harmony. It is no question of superiority or inferiority of the one sex to the other. Each sex is higher; each is lower; together they make up the perfect whole. Separate they are infirm. In co-operation they are strong. In competition they are mutually destructive. It is in the sympathetic accord of the differentiated sexes, that human progress can alone be hoped for.

“He is a half part of a blessed man,
Left to be finished by such a she,
And she a fair divided excellence,
Whose fulness of perfection lies in him.”

And blindness to this complementary relation of the sexes, so patent to Shakespeare, it is, that leads wise men—wise but ignorant, or contemptuous of biology—to sanction intersexual competition in education, and for subsistence with its ruinous effects. Men and women are constitutionally adapted to do different work in the world. To set them to do the same work is wasteful, and detrimental to the sex that is less adapted to it. It is impossible to contemplate with complacency some of the experiments in this direction, which are being carried out, and it is impossible to speculate from a medical point of view without apprehension what the outcome of such experiments may be, or what high school, and college and hall education may do for the country in a few generations, if they be pushed on with relentless zeal.

Those tall, graceful, lovely English girls, whom we see around us so plentifully to-day, and never in the world's history has woman's beauty been so beautiful as it is in England to-day—those tall, graceful, lovely girls, are the offspring of mothers who had not the advantage of a high school education. What will the next generation of English girls be like? I saw a vision once that has haunted me ever since. It was of a score of sweet girl graduates, from a celebrated college, standing together in a group on the platform of a provincial railway station, waiting for trains to carry them home at the end of the term. Sweet they were, I doubt not; most of them carried musical instruments, but they were not upon the whole, well—not just “fairest of the fair” to look upon. I am afraid I shall be called ribald and profane, but I should describe them as pantaloon-like girls, for many of them had a stooping

gait, and withered appearance, shrunk shanks, and spectacles on nose. Let us conserve the beauty of our English girls very jealously. I would rather they remained ignorant of logarithms than they lost a jot of it.

CLINICAL EVENINGS.

November 23rd, 1891.

CASE OF EXCISION OF THE ELBOW FOR INJURY.

By W. F. HASLAM, F.R.C.S.

MR. HASLAM (Birmingham) showed a man, aged 51, whose elbow he had excised nineteen months ago for a compound comminuted fracture. The injury was caused by a blow from a coupling chain that broke while he was coupling some carriages together. The outer condyle and the capitellum of the humerus were shattered, and a small piece was broken off the head of the radius, the ulna being uninjured; the soft tissues on the outer side of the joint were pulped. Excision was performed by the usual longitudinal incision, a smaller one at right angles to this being made through the damaged tissues on the outer side; the humerus was removed just above the condyles, and the head of the radius, together with the greater sigmoid cavity of the ulna, was also removed. Healing was necessarily attended by some suppuration of the bruised tissues, but the patient was able to leave the hospital at the end of five weeks. The movements of the joint are now perfect, with the exception of some slight loss of the power of extension, and the arm is for all ordinary purposes as useful as the other. Mr. Haslam drew attention to the importance of dealing promptly with these cases, and of deciding whether an attempt should be made to save the joint or whether excision should be done. He thought that, where the fracture into the joint was associated with much bruising and laceration of tissue, an excision gave a much better result than a less radical proceeding, for, in addition to the danger of an acute arthritis, there was that of a stiff joint, while with an excision these two dangers were practically eliminated. The present case illustrated

very well what result may be obtained by excision in a perfectly healthy man who has the will and determination to carry out the necessary passive movements.

A CASE OF MONILIFORM HAIRS (MONILETHRIX).*

By P. S. ABRAHAM, M.D.

DR. ABRAHAM exhibited a child $2\frac{1}{2}$ years old, the hair of whose head was Moniliform. The girl had been taken to Blackfriars Hospital in October last, and was then bald from alopecia. The short hairs present were noticed to be very brittle, and under the microscope they presented a series of nodes and internodes; there was pigment present in the nodes, and the fractures occurred in the internodes. Dr. Walter Smith, of Dublin, had first called attention to this variety of hair, and Dr. Abraham was inclined to believe that there was a bacillus at the bottom of it. Both cantharides and chrysarobin had been used, and the condition seemed to be improving.

AN EARLY CASE OF MACULAR LEPROSY.†

By P. S. ABRAHAM, M.D.

DR. ABRAHAM likewise exhibited a boy, aged 7, who was the subject of macular leprosy. Eight months ago he commenced to develop a localised erythema in Demerara; his case was at once diagnosed by Dr. Castor, and his friends sent him off by the next steamer to London. He had been treated by small doses of tuberculin, and after the third injection he had a reaction more or less typical of leprosy. It did not begin till fifteen hours after the injection was given, and it lasted for seventy hours, accompanied by great malaise, and with a distinct local reaction. He had about ten injections, and a little redness developed in the spots. After that he took 12 minims of chaulmoogra oil every day, and he had much improved, though whether the improvement was due to the

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† Published in full in the 'Westminster Hospital Reports' for 1891.

oil, to the tuberculin, to his food, or to the difference of climate it was difficult to say. On his arrival in this country there were great thickening and soft nodulation of the lobe of the right ear; a ring patch on the chest, which was discoloured, but pale in the centre; and raised erythematous patches on the cheek, elbow, and legs. Dr. Abraham excised one of the maculæ, and he showed sections of it. There was irregular cell-growth in the dermis, together with giant cells, but there were no bacilli. Several observations on the blood during the progress of the injections had been made, but no free bacilli could be found.

TWO CASES OF OPENING THE CÆCUM FOR INTESTINAL OBSTRUCTION.

By D. H. GOODSALL, F.R.C.S.

MR. D. H. GOODSALL read notes of two cases of opening the cæcum for intestinal obstruction. The first patient was a labourer, aged 46, who for eighteen months had suffered from anorexia, and was losing flesh. For six days before admission no flatus was passed, and for nine days he had been suffering from vomiting. The abdomen was resonant; there were tenderness and resistance in the right iliac fossa; enemata failed to give relief. On August 7th the cæcum was fastened to the abdominal wall; on the 9th it was opened, and 2 or 3 pints of fluid feculent matter escaped. The patient died a few hours afterwards, and at the necropsy it was found that the adhesion of the cæcum to the abdominal wall had given way, and that there had been a considerable leakage into the cavity of the peritoneum. A malignant growth was found at the commencement of the ascending colon. Mr. Goodsall then showed the second patient, who was a clerk, aged 34, on whom he had performed a somewhat similar operation in October, 1890. The bowels had been confined for nine days, and several aperients and enemata had been given, with negative results. Vomiting began two days before the operation; the abdomen was distended and resonant everywhere excepting over the cæcum. Right inguinal colotomy was performed; the cæcum was found almost gangrenous, and opened at once. In the course of an hour more than 2 gallons of feculent matter had drained away. The opened intestine was drawn well out and fixed by

stitches to the skin 1 inch from the margins of the abdominal incision. On the eighth day the congested part of the cæcum gave way externally. The patient made an uninterrupted recovery, and gained in weight for some time; since then a malignant growth could be felt above the pelvic brim, and the patient was steadily losing flesh.

CASE OF COMPLETE TRANSPOSITION OF VISCERA.

By SEYMOUR TAYLOR, M.D.

J. D——, aged 18, a stonemason, came to my out-patient room during the last summer. He complained only of trivial symptoms, and “thought that he had caught cold.” On inspecting the chest I found the heart’s apex beat to be situated on the right side, the most marked impulse being $2\frac{1}{2}$ inches below and $\frac{1}{2}$ inch internal to the right nipple. This made me suspect that there was pleural effusion on the left side, by which the heart was dislocated to the opposite side. But more careful and extended observation revealed the fact that, so far as the thorax was concerned, there was complete transposition of the viscera.

The outer edge of cardiac dulness extended from the middle of the right second intercartilaginous space downwards and outwards to the impulse in the right fifth interspace, whilst the inner margin of dulness extended from the junction of the fifth left cartilage downwards and to the right till it impinged on the apex beat.

The aortic and the pulmonic semi-lunar valves, as judged by the greatest intensity of the second sounds, are situated at the junction of the right third costal cartilage with the sternum and in the right second interspace respectively.

Similarly, without going into details of anatomical landmarks, the liver was in the left hypochondrium, whilst the spleen occupied a site in the right axillary line corresponding with what should be its normal position in the left.

And if percussion may be relied on as conclusive evidence, the cæcum occupies the left iliac fossa, and the sigmoid flexure runs from the right towards the middle line.

The area of stomach resonance was determined before food, and compared with the result of a largish meal. Before food, stomach

resonance was marked in the right hypochondrium, extending upwards to the seventh rib, outwards as far as the edge of the serratus magnus, and downwards to two inches below the costal arch. After the meal the resonant note was much diminished and did not extend below the costal arch.

It is also interesting to observe that the right testis is lower than the left.

As regards the various vessels and tubes in the body which are symmetrical, it would be somewhat tedious to recapitulate all their reversed positions, but I may be, perhaps, pardoned if I draw attention to the fact that the pulmonic ventricle, though anterior, is to the left, whilst it runs upwards and to the right. The systemic ventricle is posterior and to the right; the aortic springing from its base passes upwards towards the left side, then turns over the trachea to arch over the root of the right lung. The superior and inferior vena cava are on the left of the arterial stream, whilst the right innominate vein will cross the aortic arch from right to left.

The right recurrent laryngeal nerve would wind round the right extremity of the transverse arch, and principally supply the muscles of phonation of the right side, the left recurrent taking a course under the left subclavian artery. The ductus arteriosus would be connected with the right division of the pulmonary artery. The right lung would be bilobed and hollowed out for the reception of the heart, whilst the left lung would be trilobed.

In the abdomen the right renal vein, crossing superficially to the aorta, would have the right spermatic vein a tributary to it at a right angle. The inferior cava would perforate the left and middle leaflet of the trifolium.

In order to save the Society from the recapitulation of further anatomical details, which might be tedious, I have prepared a diagram, which shows the relative positions of the principal vessels of the thorax and abdomen. Further, the diagram has this advantage, it allows, when reversed and held up to the light, the observer to see the normal courses and situations of the transposed vessels and viscera. All the vessels and organs retain their relative positions to each other; but in this reversed position they may be compared to a mirror image, or to an etched copper-plate which reverses the original picture.

Remarks.—The majority of cases recorded are those of adult men. Many cases have been noted in which the transposition was



Diagram showing the relative positions of the principal thoracic and abdominal vessels and nerves.

- | | |
|---|---------------------------|
| 1. Systemic ventricle. | 8. Inferior vena cava. |
| 2. Pulmonic " " | 9. Pulmonary artery. |
| 3. Arch of aorta (curving over root of right lung). | 10. Ductus arteriosus. |
| 4. Abdominal aorta. | 11. Right spermatic vein. |
| 5. Innominate artery. | 12. Left " " |
| 6. Right common carotid artery. | 13. Right vagus nerve. |
| 7. Superior vena cava. | 14. Left " " |

incomplete, involving some of the viscera and their accompanying vessels only, the heart and liver being the most frequently dislocated.

The cases of complete transposition are somewhat rarer, and these are of greater interest as bearing on the subject of twin gestation.

I may at once say, however, that there is no history of the

patient having had a twin brother. But this does not preclude the possibility, or even probability, of such an event. As pointed out by Dr. Hadden, in the 'Lancet,' vol. ii, 1890, there may have been the counterpart of the divided ovum, which had withered, shrunk, and become so flattened out as to escape observation at the time of parturition. And if this be so, the sex of the blighted half must have been a male, seeing that the theory which obtains most credence in these cases is that they spring from one ovum and primitive streak, which had in the early fission stages of development this process carried to such an extent as to absolutely divide the germ into two distinct separate, yet similar halves.

It appears to me most probable that Allen Thomson's explanation of these cases is at the same time correct and scientific. His contention is that in early development the cranio-vertebral axis is placed prone on the upper part of the yolk membranes; then follows a twist on the long axis of the head and thorax of 45 degrees to the left; the abdomen follows suit till the left side of the embryo is towards the surface of the yolk.

The viscera of the two individuals of a homologous monster form one complete whole; the two germs lie side by side in the germ area of a single yolk, both prone, but still sufficiently apart to form two cranio-vertebral axes and two visceral cavities. The one to the left (looking from above) rotates to the left and is natural, the one to the right rotates to the right and is transposed. Or looked at from below, or the yolk surface, the right-hand one is natural, and the left-hand one is transposed.

In the case which I bring before the Society, the organs are healthy: there is nothing redundant or in excess, and nothing deficient.

The abnormal positions of his internals exert no baneful influence, and the condition is not incompatible with health and longevity, as cases are recorded of men who have been passed into the army and who have served as good soldiers.

As Moxon pointed out, in a case which he recorded in the 'Lancet' of 1876, transposed viscera might, unless detected beforehand, present difficulties both to the surgeon and to the physician, for example, in the operations of colotomy, gastrostomy, or in pleural effusions, or in case of syncope.

It would be interesting to enquire whether in the case of double monsters united at the umbilicus, as in the Siamese twins, the

viscera were transposed; if so, I imagine that the normal condition would obtain in that individual who was to the left, and transposition occur in the one who was to the right.

In conclusion, I may observe that the patient is not left-handed; and whilst I have failed to obtain any trace or knowledge of hereditary predisposition to similar abnormal conditions in his family, still the question of heredity is one of extreme interest.

A CASE OF NASO-PHARYNGEAL POLYPUS IN A GIRL; REMOVAL BY THE SNARE AND SPRING-CATCH FORCEPS.

By W. SPENCER WATSON, F.R.C.S.

MARY G——, a tall, thin, anæmic girl of 16½ years of age, was brought to me at the Great Northern Hospital early in October. A gelatinous polypus was clearly visible through the mouth, hanging down behind the uvula. The girl had been aware of this growth for about two and a half years, and had been operated on by Dr. Jago, of Arundel Square, who has from time to time removed small portions of it, the last operation having been performed six months ago. The relief after each operation has been only temporary, but the inconvenience from the presence of the growth has never been very great. The girl has been subject to headaches and faintness. Not unfrequently she has actually fainted. She has a slight cough and easily takes cold. She wakes up often at night almost suffocated, and with fits of choking, but never has any difficulty of swallowing. After the operations she has sometimes had no unpleasant symptoms for about three months at a stretch.

Present condition, October 1st.—A rounded, shining, greyish-white mass is seen behind the uvula, and occupying a small space on each side of it. Examined by the finger this tumour seems firmer than ordinary gelatinous polypi, but not so firm as a fibrous tumour.

Examined from the front, the left nostril is seen to be occluded far back by a rounded gelatinous polypus. The right nostril is quite clear, but the patient states that it also is often obstructed. Posterior rhinoscopy reveals a yellowish-grey, rounded mass, occupying the whole of the naso-pharynx, and completely shutting off all view of the choanæ.

Examined a week later (October 8th) the growth had shrunk perceptibly, so that it was not visible through the mouth, and the girl said she felt nothing of it. Anterior rhinoscopy and digital palpation, however, showed that the growth was still *in situ*, though lying more horizontally than before.

October 8th.—Having applied cocaine (20 per cent. solution) I at first made an attempt to catch the polypus in the loop of Jarvis's snare. This failed. A similar attempt with Krause's snare also failed.

I then passed my canula-forceps through the left nostril, and steadying the polypus in the pharynx by the left forefinger, succeeded, as I thought, in tearing away the pedicle. Suddenly, while my forefinger was still in the pharynx, the tumour disappeared, and I concluded it must have been swallowed.

A week later, however, a similar but smaller growth was seen to be occupying the post-palatine space.

On October 22nd I again applied a 20 per cent. solution of cocaine very freely to the palate, pharynx, and left nostril.

I then fixed the palate forward by the use of White's palate-hook and passed a cold-wire snare through the left nostril into the pharynx and thence into the mouth, where the loop was caught and drawn forwards.

The curved end of the pharyngeal catch-forceps (here shown) was now passed through the wire loop and the lower end of the polypus secured, Mr. Calthrop, the house physician, holding it in position.

This manœuvre enabled me to slip the loop up towards the attached part of the polypus and into the nostril, and so on to its pedicle.

The catch-forceps was now let go, the wire loop tightened on the pedicle, and separation easily effected.

The severed pedicle, however, was still held by the tightened snare, and was drawn out through the left nostril.

The growth presents an irregular oval form, having in its extreme diameters $2\frac{1}{4}$ ins. and 1 in. with a narrow pedicle, and being much lobulated. It has all the naked eye characteristics of an ordinary gelatinous or myxomatous polypus. Probably, with the piece that I removed at the first operation, the size would have been about double that represented in the figure on page 446.

The patient has had much relief and now breathes freely with

either nostril, and sleeps soundly. Her colour and aspect have much improved.



Polypus removed from naso-pharynx of a girl aged $16\frac{1}{2}$ years (actual size, from a sketch by Mr. George Spencer Watson).

The points of interest in the case are:—*a*. The unusual size and situation of the polypus. If I am right in my explanation of the disappearance of the principal bulk of the polypus in my first operation, its total length must have been between $3\frac{1}{2}$ and $4\frac{1}{2}$ inches. This is certainly very large, and the more so, occurring as it did in a young girl. *b*. The operation by which the growth was ultimately removed was, in my opinion, a great improvement on the methods often adopted, inasmuch as the method of fixing the growth by the specially contrived pharyngeal catch-forceps gives much greater steadiness and certainty in applying the loop to the pedicle. The snare used is also somewhat novel, though I have myself used it occasionally for many years. It has the merit of simplicity, and can be easily tightened by one hand, whereas Jarvis's and many other snares require the use of both the hands. The polypus in this case was fortunately easily detached, and had a narrow pedicle, and had it been of the fibrous variety or broad based, an entirely different method would have been necessary. In any case, however, I think the pharyngeal forceps would prove of essential service in these operations behind the soft palate. It has just sufficient curve to allow it to reach high up behind the palate and even to pass into one or other of the choanal apertures, and its small bulk enables us to pass it even when the space available is very limited. With the ordinary Vulsellum forceps the breadth of the blades is often too great, and there is some difficulty in guiding them exactly to the part that it is desired to seize.

The point of attachment of the polypus could not be ascertained either by anterior or posterior rhinoscopy. The view of the posterior apertures was perfectly clear, and no trace of any cicatrix or ruptured pedicle could be made out when the rhinoscope was used a few days after the extraction of the polypus.

January 25th, 1892.

TWO CASES OF COMPOUND FRACTURE OF THE SKULL IN CHILDREN TREATED BY TREPHINING.

By JOHN H. MORGAN, F.R.C.S., M.A. Oxon.

MR. J. H. MORGAN brought two children who had suffered compound fracture of the skull. The first was a boy, aged 2 years, who fell from a window on the third storey, alighting on his head. He was admitted within a few minutes to Charing Cross Hospital. He was conscious, there was no paralysis, but the pupils were dilated. The back of the head appeared flattened. From one parietal eminence to the other extended a large hæmatoma, at the extremities of which were two minute punctures. On pressing this swelling, clots mixed with brain substance exuded from each. He was immediately anæsthetised, and an incision carried from one puncture to the other over the vertex. A double line of fracture was seen running from one parietal bone to the other, and extending on each side beyond the end of the incision. At the apex of the right parietal bone was a detached piece, which was depressed, and had penetrated the brain. From both extremities of the fracture brain matter exuded. A trephine was used, and the depressed piece was elevated. Two or three other pieces of bone which were detached were lifted out, and a stream of warm boracic lotion was forcibly injected, and altogether about a drachm and a half of brain tissue was washed out. The pieces of bone were kept in warm boracic lotion, were replaced as nearly as possible in their natural positions, and the pericranium brought over them and united with catgut sutures. A small drainage-tube

was inserted at the right extremity of the wound. Except that a considerable amount of swelling of the forehead took place from extravasated blood, the child had no bad symptoms, and the greater part of the wound was healed on the ninth day. Granulations covered the two extremities of the wound, and except for an exanthematous rash on the fifteenth day, the boy had no drawbacks. One minute crumb of bone came away from the wound over the left parietal bone. The accident happened a year ago, and the boy had had no headache, no fits, and the scars showed no depression nor any pulsation.

Mr. MORGAN showed a second case, that of a boy, aged 7, who was knocked over by a Hansom cab. He was conscious when admitted. Blood and brain substance were found on his collar. There was a scalp wound with jagged edges about 3 inches long over the right parietal, not exposing the bone. On the left side was an irregular wound reaching from behind the ear backwards towards the occipital protuberance, just above the situation of the superior curved line. As this exposed a fracture, an incision was made at right angles to the wound and running obliquely upwards towards the lamdoid suture. It was then seen that the squamo-parietal suture was separated, and a fracture was detected running across the upper part of the parietal bone from the lamdoid suture behind to the coronal in front. A large area of the parietal bone was depressed, and from the interval at the squamo-parietal suture brain substance and blood were oozing. The depression not being great, no attempt was made to elevate, but the parts were syringed with a forcible injection of boracic acid lotion, the pericranium adjusted, and the scalp wound united. The amount of brain substance lost was considerable, but could not be estimated. Every precaution was taken to render the wounds aseptic, but owing to the amount of dirt which had been ground in, that on the right side suppurated. The other healed directly, but about a month after leaving the hospital a small superficial abscess formed in the neighbourhood of the wound, which healed rapidly after being incised. The boy had remained perfectly well, and had had no fits.

CASE OF EARLY BRONCHIECTASIS.

By J. WALTER CARR, M.D.

DR. WALTER CARR showed a boy, aged 8 years, who had suffered from winter cough since birth; the cough had continued also during the summer for the last two or three years, and had lately become distinctly paroxysmal. He had had measles and whooping cough about three years ago, but the cough had not been worse afterwards.

No expectoration, no foetor of breath, and no sickness with the cough, but breath very short on exertion, and slight cyanosis at times. No clubbing of fingers. Boy well nourished. Heart normal. No sign of emphysema, the limits of cardiac and of hepatic dulness being normal. No deficient resonance over the lungs, but well-marked gurgling and sub-crepitant râles over both posterior bases, the gurgling character being especially marked at the left base.

SPECIMEN OF LUNG, SHOWING ADVANCED BRONCHIECTASIS.

By J. WALTER CARR, M.D.

DR. WALTER CARR showed the lungs from a girl just 3 years old at death. She had suffered from cough for twelve months, apparently not due to measles or whooping cough, and in June, 1891, was treated for three weeks in the Victoria Hospital for Children, Chelsea, as a case of phthisis; but with one exception, during this time, the temperature was normal or sub-normal; she afterwards attended as an out-patient.

The child wasted a good deal; no clubbing of fingers; she had markedly spasmodic and choking cough, no sickness, but some foetid expectoration, and always very foetid breath. No hæmoptysis.

The physical signs were dulness at right posterior base, with very abundant coarse gurgling râles all over the right lower lobe, but varying in number from time to time: also some signs of general bronchitis.

She died very suddenly on January 19th, 1892, from pneumothorax of the left side, starting from a perforation in the upper lobe, but no marked changes, except a few adhesions, were found in this lung. In the right lung the upper lobe was in a state of grey hepatization. The middle and lower lobes were very adherent to the chest wall, and consisted entirely of large cylindrically-dilated bronchial tubes, surrounded by firm fibrous tissue.

Bronchial and tracheal glands large and fleshy, but showed no signs of tubercle.

TWO CASES OF BRONCHIECTASIS.

By W. WALLIS ORD, M.D.

DR. WALLIS ORD brought forward two cases of bronchiectasis in children. The first was a girl, aged 9, who had had measles and whooping cough seven years ago. Four years ago there was dulness and tubular breathing at the base of one lung; the temperature, which was at first high, afterwards became sub-normal, and remained so. At the present time there was dulness at the left base below the scapula, with amphoric breathing, coarse râles, and pectoriloquy.

The second case was that of a boy, aged 4, who had likewise suffered from measles and whooping cough, which, in 1889, were followed by an attack of acute bronchitis of obstinate character. Six months afterwards bronchiectasis was discovered, which was at first mistaken for phthisis. The temperature was sub-normal, and the boy had put on flesh since the summer. He was, unfortunately, unable, in either case, to obtain sputum in order to demonstrate the absence of bacilli.

CASE OF PLASTIC OPERATION FOR CONTRACTURE FOLLOWING BURN.

By BERNARD PITTS, F.R.C.S.

MR. BERNARD PITTS showed a case of plastic operation for contracture following burn. The patient, a girl aged 11, was badly burnt on the left arm in December, 1890, by the upsetting of a

paraffin lamp; the burn extended over the greater part of the arm and over the upper part of the forearm. She was admitted to St. Thomas's Hospital in September, 1891, with a tense band of scar-tissue, causing flexion of the forearm at rather less than a right angle, the scar-tissue extending over the whole circumference of the forearm; the only healthy skin was at the posterior surface of the upper arm and over the posterior aspect of the elbow-joint. The elbow could be extended to a right angle, and flexed about fifteen degrees, rotation being fairly free. A month later the band in front of the elbow was divided down to the muscle and vessels, and the denser part of the tissue dissected away, a very wide gap of 5 inches being thus made. It was found impossible to completely straighten the arm, even when the superficial fibres of the biceps and of the muscles arising from the external condyle had been divided. A large flap of skin was then raised from the posterior aspect of the upper arm, glided across the gap, and secured in position with sutures. A considerable raw surface was still, however, left both above and below the flap, in addition to the large wound made by raising the flap. These raw surfaces were carefully covered by a number of parings from the skin of the thigh and legs, taken after the method of Thiersch. The grafts were cut as thin as possible with the razor, leaving the deeper structures of the skin behind and only just causing slight punctiform bleeding. The grafts varied in size from a florin to a five-shilling-piece. All these thin grafts lived, but one thicker piece, taken from the thigh and consisting of nearly the whole thickness of the skin, died. The condition of the arm was now very satisfactory, there being free movement in all directions, except some limitation of complete extension. This limitation was no doubt due to the burn having extended to the superficial part of the muscles. Thiersch's grafts were found of great value both in covering the raw surfaces above and below the glided flap, and also in covering the wound at the back of the arm. This method of skin grafting was not entirely used, because it was desirable to have a substantial flap of glided skin across the bend of the elbow, it being more supple and less likely to be irritated by movement.

CASE OF SPINAL DISEASE, PROBABLY SYRINGO-MYELIA.

By J. A. ORMEROD, M.D.

CASE.—T. W. P——; young man, aged 19; labourer; healthy, except for present complaint. Affection first noticed in right hand seven or eight years ago, viz., a feeling of pins and needles, followed by inability to close his hand properly. He states that he cannot feel when he has hold of anything. Since that time the left hand has got gradually small, but he can always, he says, feel with this hand. For a fortnight or two before he first came to the hospital (which was on December 5th, 1891) he says that “it,” meaning a quivering sensation, went up into his arms and neck, and was felt by him in his thighs and calves.

On examination the following condition was found :—

a. Muscular Atrophy.—Atrophy, well marked though not extreme, of the muscles of both *hands*, involving the thenar and hypothenar eminences, the first dorsal interossei, and probably the other interossei also, since the fingers are in the position of interosseal paralysis. *In forearms*: some wasting of the extensor surface, and rather more marked wasting of the flexor surface (excepting perhaps the pronator radii teres). The supinator longi in pretty good condition. *Upper arms*: muscles in fair condition, deltoids good. The weakness and wasting are more marked in the right than in the left limb. No fibrillar twitching has been observed.

β. Electrical Reactions. Faradism.—Muscles of left forearm and hand all react. Right forearm: all muscles react except flexors of fingers. Right hand: no reaction obtained in the intrinsic muscles of this hand. Galvanism.—Left forearm: normal reactions (rough examination). Left first dorsal interosseous gives to 4·5 ma. KSz.ASo, muscle-twitch sharp. Right forearm: normal reactions or, at most, some quantitative deficiency in the flexors (rough examination). Right first dorsal interosseous gives no reaction; but in the remaining interossei the reaction is normal (KSZ > ASZ). No reaction of degeneration has therefore been found; but a simple reduction or extinction of electro-contractility in the more deeply affected muscles.

γ. *Spinal Column*.—There is a well-marked lateral curvature of the spine, convexity to left, in the mid-dorsal region.

δ. *Sensation*.—Light *touches* are felt by him in both hands and both upper limbs generally, but perhaps less acutely in the right hand and forearm than elsewhere. *Pain* (e.g., pinching on forearms, dry faradism on forearms, hands, &c.) is felt all over the upper limbs. Faradism perhaps not felt very acutely on the hands where the skin is thick, nor on the trunk. *Heat and cold*, as tested with test-tubes containing hot and cold water. He confuses the tubes (usually calling them both warm) over the following areas:—Right hand, forearm, and arm throughout, except perhaps inner aspect of upper arm; right side of neck and lower jaw; right face (?). Left hand, left forearm extensor surface; on left forearm flexor surface, and left upper arm he is generally right.

ε. *Other circumstances*.—No trophic lesions (except the muscular atrophy). On right hand there is a scar from a burn, but this burn was not painless (about six years ago). Left pupil rather larger than right. Gait natural. In lower limbs no atrophy. Patella tendon reflexes well marked; plantar reflexes not obtained.

ζ. *Family History*.—Father died of consumption. Mother alive, healthy. Three living brothers, one living sister, suffer from coughs; one “has bronchitis.” One brother died at $1\frac{1}{2}$ years, “of consumption” (?).

On the evidence of the chronic muscular atrophy, the peculiar loss of thermal sense, and the spinal curvature, the case was presented to the Society as probably an example of syringo-myelia.

CASE OF MULTIPLE EXOSTOSES.

By W. BRUCE CLARKE, F.R.C.S.

MR. TUNNICLIFFE exhibited for Mr. Bruce Clarke a case of multiple exostoses in a man aged 36, in whom one of the osteomatous growths had developed into a sarcoma. All the exostoses stopped growing at the age of 25, but the mass in the abdomen started growing four years and a half ago, and now filled the whole of the left side of the pelvis. The inguinal glands were enlarged;

there was difficulty of defecation, and increased frequency of micturition; pain was also present along the course of the great sciatic and obturator nerves.

CASE OF PRIMARY CHANCRE OF THE CHEEK.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. MARMADUKE SHEILD showed a case of primary chancre of the cheek. The patient was a woman, aged 34, and a widow. Near the centre of the left cheek was a formidable-looking, dusky-coloured swelling the size of a florin. The edges were sharply defined, and it was devoid of discharge or ulceration. The sub-maxillary glands were much enlarged, and the skin covered with a dusky syphilide. The patient was taking mercury energetically, and the chancre had already reduced in size, while the rash had faded. The case had come under his care fourteen days ago. There was no history of infection, and all that was known was that the "sore" had existed for about two months. Mr. Sheild especially referred to an almost exactly similar case, with an illustration, published by him in the 'British Medical Journal' of February 5th, 1887.

February 22nd, 1892.

THREE CASES OF ARTHRECTOMY OF THE ELBOW-JOINT.

By H. HUGH CLUTTON, F.R.C.S.

MR. CLUTTON exhibited three cases in which he had performed arthrectomy by a new method for the relief of tubercular disease of the elbow-joint in children. During 1888 four such cases were submitted to operation at the Victoria Hospital for Children, one of which had eluded search, though when last seen the result was quite equal to that in those shown. The operation consisted in opening the joint by dividing the olecranon and removing all the

diseased structures with a sharp spoon and scissors. The parts were reunited and kept absolutely at rest. No passive movement was at any time performed, and a plaster of Paris splint was continued for three or four months. The range of movement now present was obtained entirely by the patients themselves. The results were seen to be but little short of the normal. If the cartilages at the time of operation were firmly fixed to the bone beneath and the wound kept aseptic, the joint would not ankylose, even though it be kept at rest for some months; whereas, if the cartilages be detached and a movable articulation be required, the ends of the bones must be removed as in the ordinary method of excision.

CASE OF EPITHELIOMA OF THE TONGUE.

By W. BRUCE CLARKE, F.R.C.S.

MR. BRUCE CLARKE exhibited a case of epithelioma of the tongue which had been seen in the gummatous stage, but, the patient having absented himself for many months, the disease was now too far advanced for operative interference. He proposed to inject a solution of aniline dye into the growth. Such solutions had been stated to have a beneficial effect. He hoped to be able to relate the result at a future meeting of the Society.

A CASE OF ACROMEGALY.

By SIDNEY PHILLIPS, M.D.

DR. SIDNEY PHILLIPS showed a case of acromegaly. The patient was a labourer, aged 50, who had come to St. Mary's Hospital for sciatica. He had never had gout or syphilis; he had suffered from pains, possibly rheumatic in the shoulders; in his younger days he had drunk rum rather freely; he had not been suffering from any mental depression or emotion. He could not say when the symptoms of acromegaly commenced, but he had had to wear larger boots for the last four years.

The face presented the usual elongated shape of acromegaly; the forehead low; the lower jaw much deepened. Several teeth

had been lost: there was no apparent hypertrophy of those remaining; there were thick bony prominences about the external angles of the orbits; no exophthalmos.

The alar cartilages of the nose were greatly enlarged and thickened; the cartilages of the ear did not appear enlarged; the tongue was much hypertrophied; the tonsils large; speech thick and guttural; no loss of hair from head; complexion, healthy looking.

The hands were greatly enlarged and spade-like, and the enlargement extended to the bones of the forearm; the nails were flattened and appeared thinned: they presented fine longitudinal striation, and were very brittle.

The feet were enormous and spread out like the hands.

The head appeared sunk on the shoulders, and the neck short; there was slight undue prominence of the four or five spines of the dorsal vertebræ below the 5th, forming a slight angular curvature. There was no tenderness about the spines, and they did not seem thickened; the abdomen was very prominent; the pelvic bones thickened and enlarged. No thyroid gland could be felt. There was a slight want of percussion resonance over the upper piece of the sternum.

The patient frequently suffered from headache: he sweated profusely. There was no bulimia; no loss of taste; no albuminuria; no glycosuria; and there were no hæmorrhages.

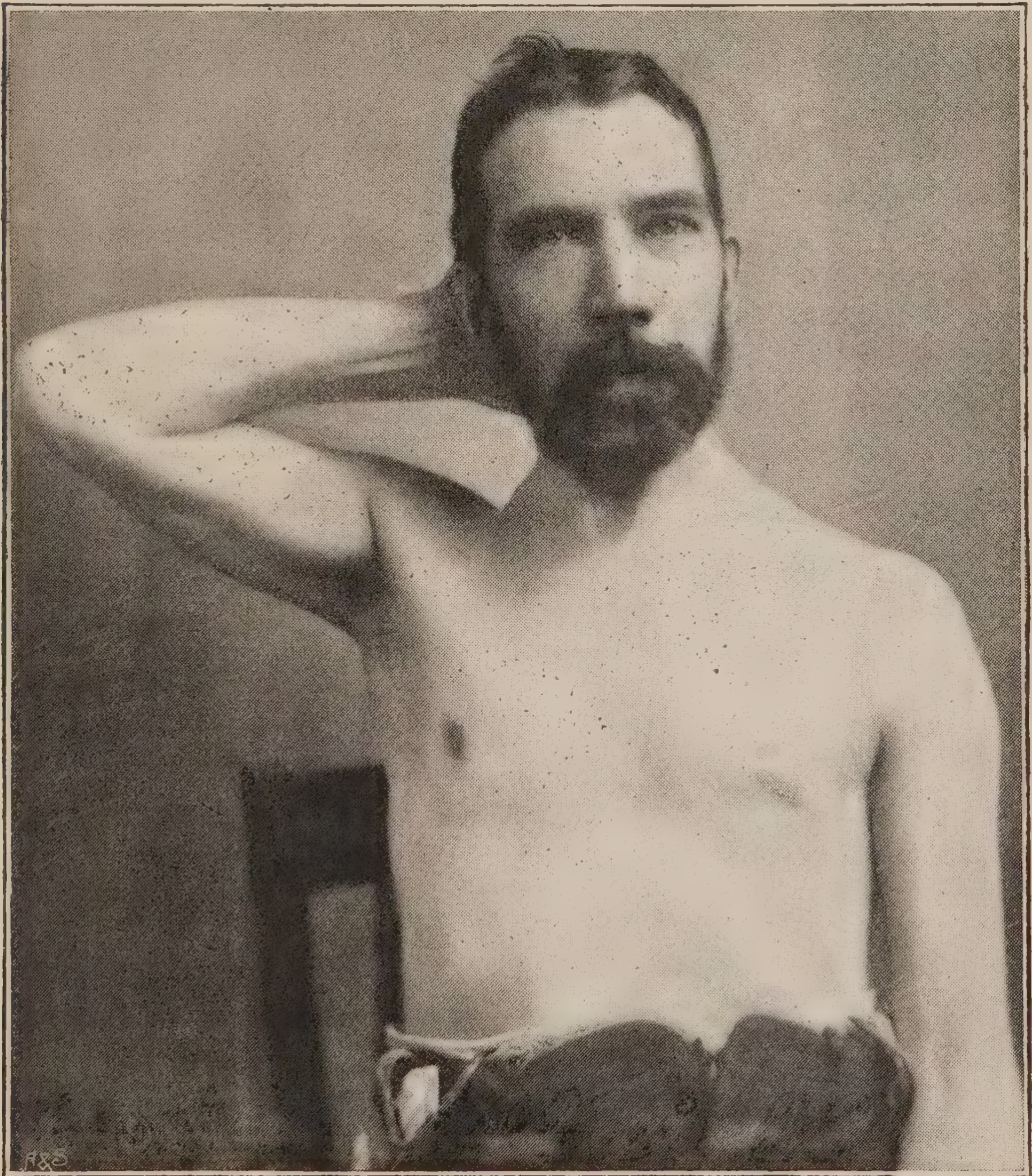
He said his sight was failing, but he could read brilliant type; no optic neuritis or atrophy, and no hemiopia.

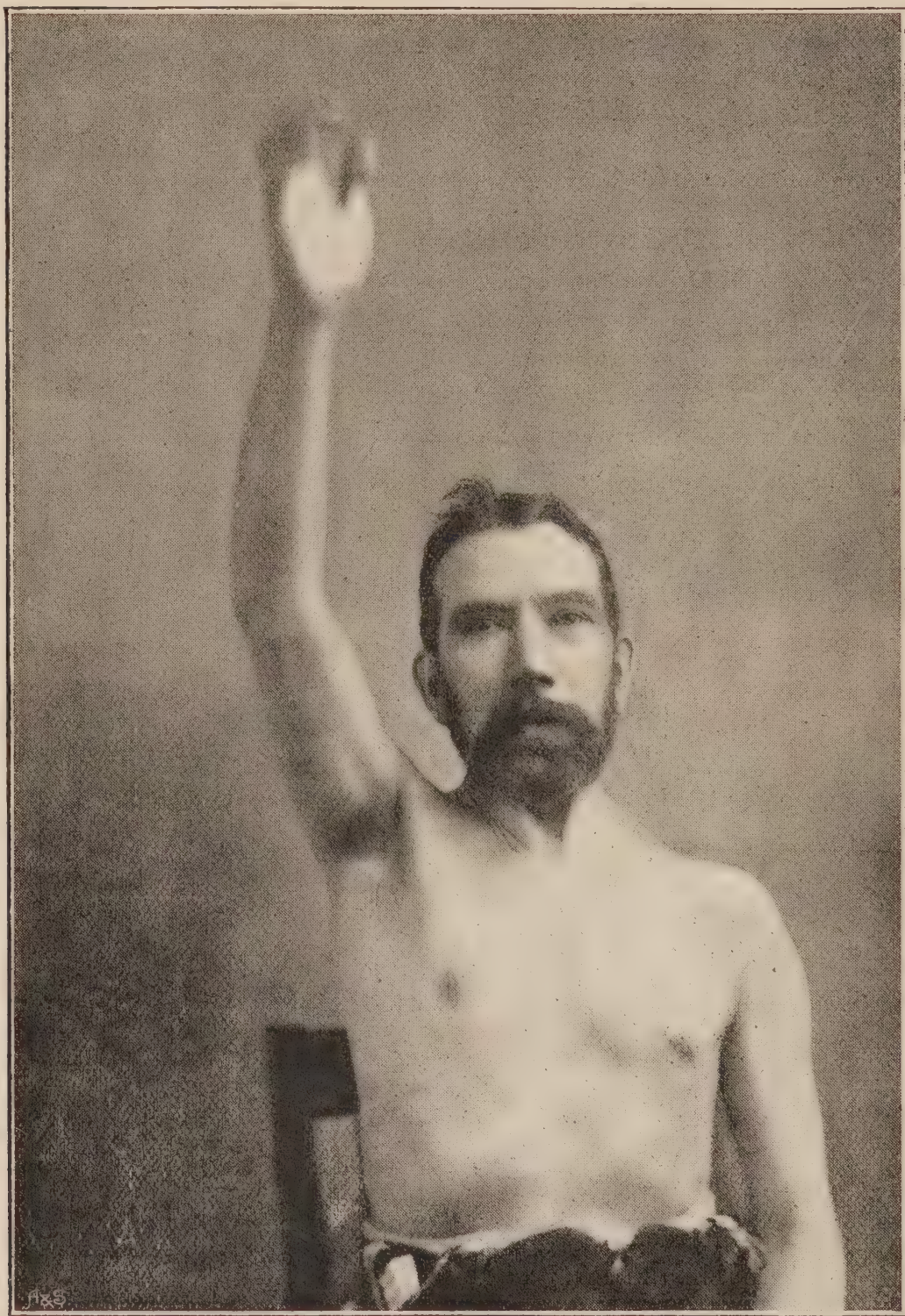
The patient was intelligent and clear in mind, and his memory good. There was no loss of power in the limbs, but he had had severe sciatica for some months, with much wasting of the thigh, muscles, and very intractable; whether this was in any way connected with the acromegaly was doubtful. The knee-jerks were present. There was a pendulous body, the size of a hazel nut, on the back of the nature of molluscum fibrosum, such as had been noticed on other patients with acromegaly.

CASE OF OPERATION FOR UNREDUCED DISLOCATION OF THE SHOULDER.

By A. PEARCE GOULD, M.S.

C. G——, aged 50, fell on February 15th, injuring his right shoulder. He was seen by a doctor next day, and was then told that he had dislocated the shoulder. The same day it was “reduced,” and the arm was bandaged to the side. The pain was incessant and severe, and on March 29th I saw him, and found the humerus dislocated below the caracoid process, and the whole





arm, forearm, and hand œdematous, paralysed, and the seat of severe pain, which was much worse at night. Chloroform was given and reduction attempted, but without success. Three days later an anæsthetic was again administered at Middlesex Hospital, and reduction attempted with the aid of pulleys, but unavailingly. I therefore admitted C. G—— to the ward, and on April 5th I cut down on to the joint along the anterior border of the deltoid muscle, as if for excision of the shoulder. After dividing the subscapularis, supraspinatus, infraspinatus, and teres minor tendons completely, with the aid of the pulleys fixed to the arm, and a respiratory used as a lever to force out the head of the humerus, I succeeded in replacing the head of the bone in the glenoid cavity. Considerable force was requisite for this, so much so that a senior colleague advised me to excise the head of the bone. The wound was closed and dressed antiseptically. It healed by first intention. The pain was immediately relieved and never returned, the œdema quickly passed off, and the muscular power was regained.

On June 2nd I broke down adhesions in the shoulder under gas. For some time the muscles supplied by the ulnar nerve remained paralysed, but they have now quite recovered. There is full movement in the joint.

In this case the dislocation was complicated with pressure upon the axillary vein and the nerves of the brachial plexus, the ulnar nerve being more seriously injured than the others. The range of movement enjoyed by the patient is shown by the woodcuts, which are copied from photographs.

CASE SHOWING THE RESULT OF OPERATION FOR OLD UNREDUCED DISLOCATION OF THE SHOULDER.

By W. WATSON CHEYNE, F.R.C.S.

MR. WATSON CHEYNE showed a second case of the above, but in which four months had elapsed between the dislocation of the bone and its restoration, the man, in addition, having failed to attend the hospital subsequently. In consequence of this and of having had to divide all the muscles with the exception of part of the

pectoralis major, as well as to clear out the glenoid cavity, range and power of movement were somewhat defective, and the head of the bone had slipped slightly forward, but the condition of the patient was much better than before the operation, and his arm was still gaining in strength.

CASE OF UNUSUAL MOBILITY OF THE SPLEEN.

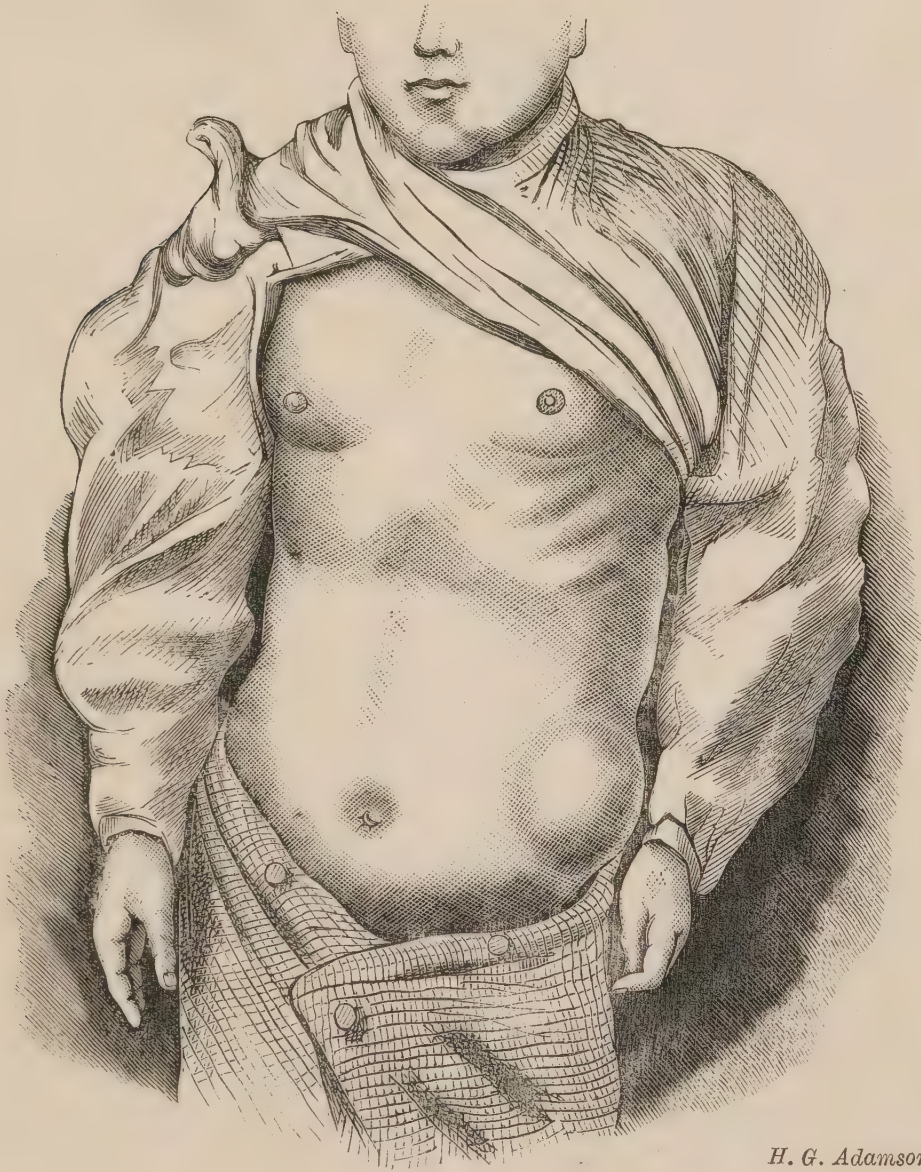
By LESLIE OGILVIE, M.B.

EDWARD W——, aged 12 years, is the third child of a family of six. The first two are healthy; the fourth died of bronchitis in



H. G. Adamson.

early infancy ; the fifth shows signs of rickets, and the spleen can be distinctly felt and is very movable. The sixth is now about four years old and looks healthy, showing no signs of rickets. The



H. G. Adamson.

mother had a miscarriage between the births of the fourth and fifth child. No history or signs of congenital syphilis can be made out, and the mother looks very healthy. The father had rheumatic fever twice, but is said to be healthy. Edward, the third child, suffered from rickets in early childhood. The dentition was slow ; he only began to walk when about three years of age ; at six " he had his legs put in splints " on account of the deformity. He was subject to bronchitis, and his belly was very large for several

years. At present he appears fairly healthy, though somewhat stunted in growth. The rickety history is shown by the enlargement of the epiphyseal extremities of the bones of the forearms, the anterior curvature of the shafts of the tibiæ, the rachitic rosary, and the flattening of the lateral portions of the ribs. His spleen can be easily grasped and moved about. He can, on straining or coughing, depress the spleen, so that it can be seen as a tumour on the left side of the abdomen, as shown in the second drawing, no such swelling being visible when he remains quiescent. It is probable that the increased weight of an enlarged spleen in infancy caused stretching of the gastro-splenic omentum, and the unusual length of the ligaments permits undue mobility.

CASE OF FRIEDREICH'S ATAXY.

By J. HUGHLINGS JACKSON, M.D., F.R.S.

DR. JAMES TAYLOR (for Dr. Hughlings Jackson) showed a young man, aged 18, exhibiting the typical symptoms of hereditary ataxy. There were present irregular tremor of head, also of hands and arms, inability to "hit" accurately with one hand a point on the opposite limb or on the face, an unsteady, lurching walk, and inability to stand steadily with the feet together, as well as inability to stand at all if the eyes were closed. The knee-jerks were absent, the pupil reflex was preserved. There was marked lateral curvature. There were also vague cramp-like pains in both legs and in the right shoulder, and a tight feeling on left side. He had slightly blurred speech, and nystagmus. The deformity of the foot was characteristic. An elder brother was the subject of the same disease, and in each patient the first symptoms came on after an attack of diphtheria nine years ago. This was, apparently, followed by diphtheritic paralysis, from which the patient made a fair recovery, and the first symptoms of the present condition followed in a few months.

March 21st, 1892.

CASE OF AN INFANT AFTER ACUTE EPIPHYSITIS.

By EDMUND OWEN, F.R.C.S.

MR. EDMUND OWEN showed a female infant of 9 months, who, last January, was admitted to the Children's Hospital with acute sub-deltoid abscess. The infant looked extremely ill; axillary temperature 101° ; pulse rapid and feeble. Under chloroform, the movements of the shoulder-joint were free; the humerus was thickened just below the tuberosities. The diagnosis made was acute septic inflammation—probably with necrosis—at the diaphysial aspect of the junction cartilage. On an incision being made down to the bone an abscess was evacuated, and a small opening was found leading into the humerus at the surgical neck. This opening was enlarged, two small sequestra were extracted, and a good deal of granulative tissue was scraped out of the expanded shell of bone. During this operation the epiphysis became detached. The cavity was washed out with a chloride of zinc solution, and temporarily drained, the upper part of the incision being closed with sutures. The hand was then fixed across the opposite side of the chest, and the arm was secured in dry dressings and a bandage, as if for separation of the epiphysis. The infant's temperature quickly dropped; the wound closed; the epiphysis once more became attached to the shaft, and the child has recovered with a movable joint and a sound limb. Mr. Owen was of opinion that this case had been on the verge of becoming one of those which were described as acute arthritis of infants; and though he could not adopt the term as a separate pathological entity, he was well aware that arthritis did often follow the epiphysitis. He was in favour of early exploration of the bone in these cases.

CASE OF INJURY TO MEDIAN NERVE; OPERATION; RESTORATION OF FUNCTION.

By T. PICKERING PICK, F.R.C.S.

MR. PICK showed a little girl, aged 12, who was admitted into St. George's Hospital, under his care, on March 25th, 1891, with the following history:—

On January 31st, 1889, whilst carrying a bottle, she fell down, and, breaking the bottle, the glass cut her left arm. She was seen at once by a medical man, who states that he sutured, under an anæsthetic, all the tendons he could find; but that he was unable to find the ends of the median nerve. The wound healed quickly, but ever since there has been loss of sensation in the thumb, index, and middle fingers, with some impairment of the movements of the thumb.

On admission, she was found to be a pale, but fairly healthy-looking girl. On the front of the left forearm, at the junction of the middle and lower third, was a cicatrix, which was slightly tender to the touch. There was absolute loss of sensation in all parts of the hand supplied by the median nerve; there was considerable wasting of the thenar eminence, and loss of the movements of the thumb. The skin of the thumb, index, and middle fingers, and, to a less extent, the skin of the radial side of the ring finger was glossy, but there was no eruption. These fingers were shrivelled, cold, and of a purplish hue. There was no loss of sensation in the little finger or on the ulnar side of the ring finger, and the movement of the muscles supplied by the ulnar nerve was perfect. The urine was normal.

On April 9th an anæsthetic was administered, and the forearm having been rendered bloodless, an incision, about 4 inches in length, was made, with its centre over the scar, parallel to, and on the ulnar side of the tendon of the flexor carpi radialis. After division of the deep fascia, the bulbous end of the divided nerve was soon found; but there was considerable difficulty in finding the lower end, and it was only by dissecting out the nerve at the lowest extremity of the incision and tracing it upwards, that the end could be discovered. It was much attenuated. After the ends of the nerve had been refreshed, it was found impossible to approximate them, even after the upper end of the nerve had been stretched, and though the hand was forcibly flexed at the wrist. The ends still remained separated to the extent of about an inch. Lying alongside the upper fragment, and adhering to it, was a mass of what was apparently condensed connective tissue, which had probably been formed by inflammatory exudation from the sheath of the nerve. This was dissected away from the nerve to within a quarter of an inch of its lower end, by which it was allowed to remain attached to the end of the nerve; it was turned downwards, and

sutured to the lower end of the divided nerve by two sutures. The two ends of the nerve were thus connected together by about an inch of scar tissue. The wound was now closed by a continuous suture and dressed.

After the operation the case went on well; there was no rise of temperature, and, on the 20th (eleventh day), the wound was dressed, and found to be healed. There was thought to be some slight return of sensation in the fingers. She was discharged on the 29th, with instructions to show herself occasionally.

She did not present herself again until October, seven months after the operation. It was then found that sensation had returned in the parts supplied by the nerve; the fingers had assumed a much more natural aspect, but the muscles of the thenar region were still somewhat wasted.

CASE OF SENILE TUBERCULOSIS OF THE SKIN.

By T. COLCOTT FOX, M.B.

DR. COLCOTT FOX introduced a woman, aged 66, who was the subject of senile tuberculosis of the skin. For six or seven years she had had a lupus condition of the skin of the back of the hand, which had of late assumed a secondary papillomatous character. She had originally struck her hand and inflicted a wound at the time she was nursing her husband and daughter, both of whom died of phthisis. He exhibited drawings of two other cases, one in a woman of 72 and the other in a man aged 82, in both of whom the lesion was on the back of the hand. In two of the cases the parts examined microscopically presented all the characters of tuberculosis, except that bacilli were not found.

CASE OF NEPHRECTOMY.

By C. B. LOCKWOOD, F.R.C.S.

MR. LOCKWOOD showed a healthy-looking woman on whom he had performed nephrectomy. She had suffered from renal symptoms for the last fifteen years, which had been bad for the last seven. She came under his care in November, 1891, with a loin tumour, pain on micturition, cystitis, and pain in the lumbar

region. She was passing 30 to 60 ozs. of urine per diem, which was acid, and contained one-sixth pus. He cut down by a lumbar incision, and found a pyo-nephritic kidney, which he had removed. On opening it a large calculus was found, which could not be felt, and which was only discovered on making a section of the organ.

COMPOUND, COMMINUTED, DEPRESSED FRACTURE OF THE SKULL TREATED BY TREPHINING AND REPLACEMENT OF BONE.

By W. H. BATTLE, F.R.C.S.

MR. BATTLE exhibited a girl, aged 5, who, on February 6th, 1892, slipped off the pavement and became crushed between the wheel of a van and the kerb. On examination there was found an irregular fracture of the skull with considerable depression of bone, the latter being comminuted, and the area depressed measuring 2 ins. by 1 in. Trephining was performed, the depressed portions elevated and left in position, and the piece removed by the trephine replaced. The pericranium was united by silk sutures, and the wound completely closed, no drainage-tube being used. Perchloride of mercury solutions and dressings were employed, and the damaged edges of the wound excised. The patient made a good recovery in spite of the fact that she also suffered from a green-stick fracture of the right clavicle, a compound comminuted fracture of the right tibia, a compound fracture of the right fibula, and an extravasation of blood into the right iliac fossa. As a result the vault of the skull was completely restored to its proper shape, strength, and feel.

RESULT OF PARTIAL REMOVAL OF THE LEFT CLAVICLE FOR NECROSIS.

By W. H. BATTLE, F.R.C.S.

MR. BATTLE likewise showed a woman, aged 34, who, in 1888, underwent an operation for necrosis of the clavicle; a great deal of the bone was removed. The disease was of syphilitic nature, and a large gummatous ulcer, which remained over the part when she

first came under his observation, healed under iodide of potassium. There was one-third of the clavicle wanting, and there was no attempt at new formation of bone, there being merely a strong fibrous union between the ends of the fragments. Notwithstanding this, it was surprising what good power over the upper extremity the patient possessed, she being able to lift the hand above the head, and do her back hair.

CASE OF SPINA BIFIDA OCCULTA: NECROSIS OF FOOT AND TALIPES.

By A. MARMADUKE SHEILD, F.R.C.S.

A BOOT-MAKER, aged 23, was first seen by me on December 1, 1891. He had lately been an inmate of the National Orthopædic Hospital, under the care of Mr. Fisher, for "Talipes equinovarus" in both feet. This had been "coming on badly" for two years, but had existed to a minor degree since birth, and owing to the increasing difficulty in locomotion he had adopted the sedentary life of a shoe-maker. His feet were treated by manipulations and tenotomy with excellent result, but bad corns had formed under the fifth metatarso-phalangeal joint, and, on the left side, a perforating ulcer existed, leading to carious bone. The muscles of both legs were wasted and flabby, the bones well developed, there were none of the ordinary signs of ataxia present. The knee-jerks were rather strongly developed. The feet sweated excessively, but there was no marked anæsthesia of the skin. A typical perforating ulcer was seen under the fifth metatarso-phalangeal joint, and the probe reached carious bone. On examining the spine a slight depression was observed near the lower lumbar region, and here the arches of the vertebræ were markedly absent, so that the fingers sank into the spinal canal. A plentiful growth of dark hair was observed in this situation also. The bladder acted well, but the sphincter ani was weak in its action, so that if the patient had diarrhœa, he was unable to restrain his evacuations. The electrical reactions of the muscles of the lower extremities had not been tested. His mother stated that when pregnant she fell and struck her back, and placed her hand on the identical place where her son suffered. What was more to the point was the fact

that the doctor who confined her noticed the condition, and said "he could lay his thumb in the child's spine, but that nothing could be done." The mother also stated in her note an interesting point that she thought the child was like others, excepting for the perpetual state of relaxation of the bowels.

On December 3rd I cut out the ulcer and corn freely, and by a lateral incision, resected the extensively carious ends of the metatarsal bone and phalanx of the fifth toe. The parts healed soundly, and the patient was discharged on the 31st December.

I have ventured to apply the term "*spina bifida occulta*" to this case, for it closely resembles in its symptoms the interesting group of cases collated and related by Mr. Sutton, in the '*Lancet*' of 1887. I would not venture to explain the exact pathological condition in the present case of the lower part of the spinal canal and cord. But I would especially point out that in several of the cases related by Mr. Sutton, the association of talipes with necrosis of the bones of the foot was observed, and an overgrowth of hair was apparent over the lumbar spine.

April 11th, 1892.

CASE OF CONGENITAL UMBILICAL FÆCAL FISTULA.

By W. WATSON CHEYNE, F.R.C.S.

MR. WATSON CHEYNE brought a male child, aged 3 months, suffering from congenital umbilical fæcal fistula. It appeared to be an instance in which the bowel ended at the umbilicus. An anus was present, and a catheter passed up it for an inch and a half. A catheter passed in at the umbilicus only appeared to take an upward direction. He imagined that it was an instance of persistent pervious Meckel's diverticulum, and he thought that though an operation to connect the gut with the rectum was feasible, yet the child would not be strong enough to survive it. (The child died about three weeks later, and it was found that the gut below the fistulous opening in the lower part of the ilium was present and patent, but was extremely narrow, only admitting a small catheter.)

FACIAL CHANCRE.

By W. HARRISON CRIPPS, F.R.C.S.

MR. HARRISON CRIPPS exhibited a case of facial chancre. The patient, a middle-aged man, presented himself at the St. Bartholomew's skin department for a growth between the lip and chin. The size at that time was that of a florin. The growth was raised a sixth of an inch above the skin level, the edges being abrupt; its surface consisted of hundreds of papillæ about the eighth of an inch in length, looking like a warty epithelioma. The surface was dry and not excoriated. There was no discharge. The history given by the patient was that he was perfectly well ten weeks ago; he was then cut by the barber whilst being shaved. Between two and three weeks later he noticed the growth first made its appearance; in about three weeks it had attained its present size. A fortnight later a rash appeared on his body, with sore throat. The patient was now covered with a most typical secondary squamous rash. The interest in the case was the singular appearance of the primary sore, which had no resemblance to the ordinary Hunterian chancre, looking much more like an epithelioma or warty growth. Had it not been for the history of the case and the secondary squamous rash the diagnosis would have been very difficult.

AMPUTATION OF THE ENTIRE PENIS BY
THIERSCH'S METHOD.

By W. HARRISON CRIPPS, F.R.C.S.

THE patient, a man of 40, had cancerous ulceration of the penis, engrafted upon the scar of an old syphilitic ulcer. The disease, of eight months' duration, had destroyed the organ down to the level of the scrotum, the base of the penis being represented by a hard fungoid mass. A semicircular incision was made round this, and carried through the raphe of the scrotum, dividing it into two halves. The corpora cavernosa were dissected off the pubic bone, and cut completely away. The urethra was dissected downwards for an inch and a half, and brought out through a buttonhole slit made on the lower part of the perineum an inch in front of the

anus. It was then cut off level with the skin, slit open for a short distance, and stitched to the skin. The patient made a rapid recovery, and when shown to the Society the urethral opening, which would admit a full-sized catheter, could be seen a little in front of the anus. He had perfect control over the urine, and could urinate either in the sitting or standing position with freedom and comfort. Mr. Cripps alluded to the papers of Mr. Wheelhouse, Sir W. MacCormac, and Mr. Pearce Gould on this operation, and considered that there was much advantage in bringing the urethra out so near the anus, for it prevented the patient wetting himself on going to the closet.

INGUINAL COLOTOMY (ONE YEAR AND A HALF AFTER OPERATION).

By W. HARRISON CRIPPS, F.R.C.S.

MR. HARRISON CRIPPS exhibited a patient in whom inguinal colotomy had been performed nearly a year and a half ago for rectal cancer. The patient had markedly gained weight and strength since the operation, and was able to go through his daily avocations without discomfort or trouble. As a rule the bowels acted once a day.

CASE OF PERIPHERAL NEURITIS FOLLOWING INFLUENZA.

By J. MITCHELL BRUCE, M.D.

IN January, 1890, a patient, a single woman of 20 years, had influenza, but did not keep her bed. The following July she complained of pain in the right sub-axilla, which was called "intercostal neuralgia." Twelve months later, in July, 1891, she had pain in the right shoulder, then in the right thumb, followed by trophic change in the right thumb nail, and these have persisted ever since. Evulsion of the thumb nail was performed in September, 1891.

She now complains of pain in right upper limb, in the thumb, and second and third fingers. The skin of fingers and part of hand is glossy; the thumb nail shows trophic changes; there is marked

muscular weakness, limited to the thumb. There is no anæsthesia, but disturbed and greatly increased sensibility to contact in thumb, and possibly in parts of the limb. Along most of the right nerve-trunks, especially radial and musculo-spiral, there is tenderness on pressure. This is also marked in right axilla and over clavicle. On points of fingers—especially thumb-tip, second knuckle, and third knuckle, there are certain tender spots; and over the right chest tenderness is also present in the area corresponding to probably the 10th intercostal nerve, and also para-spinal. Pain is complained of in many parts of right upper limb, below the distribution of the descending branches of the cervical plexus, and inferiorly involving the thumb and second and third fingers posteriorly down to terminal articulation. No eruption has ever appeared; there has been no atrophy of muscles, nor electrical disturbance.

CASE OF SYPHILITIC DACTYLITIS IN AN INFANT.

By T. COLCOTT FOX, M.B.

DR. T. COLCOTT FOX showed an infant, aged 5 months, with symmetrical enlargement of the metacarpal bones of the thumbs and of the metatarsal bones of the great toes. The child, a first, and reputed a seven months' one, had been under mercurial treatment for some weeks for a wide-spread syphilide and "pseudo-paralysis" of the legs and left arm. Dr. Fox said thickening of the long bones, and Wegner's osteo-chondritis with pseudo-paralysis were frequently met with. The enlargement of the hand and foot bones was much less frequent, especially of the phalanges. He had elsewhere, however, described two cases of syphilitic dactylitis in infants. The diagnosis from tuberculosis was sometimes difficult.

The enlargement in this case was probably due to an osteomyelitis.

LUPUS ERYTHEMATOSUS DISSEMINATUS.

By T. COLCOTT FOX, M.B.

DR. FOX likewise introduced a woman, aged 31, with lupus erythematosus of the face, ears, and scalp, and also discs dis-

seminated thickly over the trunk and upper arms. She had hip disease at three years old; the lupus began on the face at about 17 years, and the trunk had been affected for about six months. The patient's father died of phthisis, and her child had suppurating neck glands. Dr. Fox pointed out the rarity of these disseminated cases, and their interest from a diagnostic and etiological point of view. Kaposi, Boeck, and Hallopeau had described malignant or febrile exanthematic cases. No such attack had so far been observed in the case shown.

CASE OF TUMOUR OF THE HEAD OF THE HUMERUS.

By A. MARMADUKE SHEILD, F.R.C.S.

MR. SHEILD showed a case of tumour of the head of the humerus, which occurred in a girl who had suffered from obscure pains in the right shoulder for six years. The swelling appeared to be an osteoma, and caused some displacement of the head of the bone.

A SPECIMEN OF PLUGGING OF THE DUODENUM FROM A LAMB.

By the PRESIDENT (JONATHAN HUTCHINSON, F.R.S.).

THE PRESIDENT exhibited a mass, 3 inches long, formed of fragments of undigested food, which had acted as a plug to block the duodenum of a lamb between two and three months old, which, owing to the death of its mother, had suddenly been thrown on its own resources to obtain food, and had chosen its diet injudiciously.

PICTURE OF A CASE OF INFECTIVE OR MELANOTIC FRECKLES IN SENILITY.

By the PRESIDENT (JONATHAN HUTCHINSON, F.R.S.).

THE PRESIDENT also exhibited the picture of a case of the above. It began with freckling—often symmetrical—of the eyelids, and

then on one side the pigmentation spread. The mucous membrane and cornea were also affected, which made the case unique. He feared it might end in malignancy, for he had twice seen epithelioma develop in connexion with similar cases.

AN APPLIANCE FOR FACILITATING THE PERFORMANCE OF MULES' OPERATION.

By R. BRUDENELL CARTER, F.R.C.S.

MR. BRUDENELL CARTER exhibited an appliance for facilitating the performance of Mules' operation. This consisted of a small hollow ball of india-rubber, furnished with a stopcock, connected by a short tube with a detachable syringe, and capable of being quickly distended by air. As soon as the contents of the sclera were completely removed the ball was inserted, and was sufficiently filled with air accurately to occupy the cavity. The pressure thus exerted speedily arrested bleeding from the divided vessels; and as soon as this was done the ball was suffered to collapse by opening the stopcock, and was removed to make way for the glass globe, the insertion of which completed the operation. Before contriving the india-rubber ball, Mr. Carter was accustomed to fill the scleral cavity with pieces of sponge, which were tedious to insert and to remove, and which were far less efficient as a means of arresting bleeding by compression.

SPECIMEN OF UNREDUCED SUB-CLAVICULAR DISLOCATION OF HUMERUS.

By T. F. HUGH SMITH, F.R.C.S.

Abstract of the Clinical History and Pathology of the Case.

MARY ANN C —, aged 69. As far as could be ascertained, the injury, resulting in dislocation, occurred at end of October, 1889.

The dislocation was first recognised in the end of April, 1890, but no attempt was then made at reduction.

On June 6th, 1890, patient admitted under care of Mr. Boyce Barrow, Royal Free Hospital.

The notes furnished by Registrar are as follows:—"In con-

sequence of her age, and the fact that she possessed good movement of the arm, no operation was performed, and she was discharged on the 19th of June, 1890."

Death occurred from senile decay, accelerated by gangrene of feet, on November 7th, 1891. (Dissection made by Mr. J. H. Targett, F.R.C.S.)

Pathology.—Head of humerus lies in subscapular fossa, $2\frac{1}{2}$ inches internal to coracoid process; subscapularis muscle relaxed and atrophied.

The long head of biceps retains its position in bicipital groove, as far up as anatomical neck, at which point it turns abruptly outwards at a right angle, working over a pulley-like structure.

The glenoid fossa appears to be normal, and the capsule of the false joint is made up of the anterior portion of the true capsule and the expanded tendon of subscapularis muscle.

[N.B. The specimen is now in the Museum of the Royal College of Surgeons, England.]

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